THE PLANT DISEASE REPORTER

Issued By

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 44 **Number** 11

November 15, 1960

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

PLANT DISEASE REPORTER
Epidemiology Investigations, Crops Protection Research Branch
Plant Industry Station, Beltsville, Maryland

THE PLANT DISEASE REPORTER

Crops Research Division
Volume 44

Plant Industry Station, Beltsville, Maryland
November 15, 1960
Number 11

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THE SUSCEPTIBILITY OF PIGWEED TO RHIZOCTONIA SOLANI IN IRRIGATED FIELDS OF WESTERN NEBRASKA[1]

M. G. Boosalis[2] and A. L. Scharen[3]

Summary

Results from this study show that pigweed, Amaranthus retroflexus, is susceptible to at least 15 physiologic races of Rhizoctonia solani. The majority of diseased pigweeds from the sugar beet and alfalfa fields were infected with only one physiologic race of R. solani. However, some of the pigweeds from these fields yielded 2 and 3 races. Four races were isolated from a few individual weeds from the alfalfa field. This soil-inhabiting fungus caused lesions on the roots and/or stems of pigweeds. Damping-off of pigweed was not uncommon in the alfalfa field. Results from greenhouse tests showed that irrigated crops such as bean, sugar beet, alfalfa and potato were susceptible to one or more of the races of R. solani from pigweed. Some of the races were not pathogenic to any of the above crops. All of the races, however, were pathogenic to pigweed. Nearly all of the races isolated from naturally infected pigweeds were recovered from diseased roots and stems of this host buried in soil 1 year. Furthermore, the buried tissues of this weed yielded several races in addition to those isolated from the intact, diseased roots and stems.

Results from field surveys in irrigated fields of sugar beet and alfalfa in the vicinity of Scottsbluff, Nebraska indicated that many of the pigweeds, Amaranthus retroflexus, were diseased. Examination of the roots and basal part of the stems of these weeds revealed the presence of dark brown lesions typical of those incited by Rhizoctonia solani Kuehn. Furthermore, mycelia similar to that of R. solani were discerned within and on the diseased tissues of pigweeds from these fields. Schuster, et al. (3) previously reported the isolation of one culture of R. solani from the root of pigweed. The authors concluded that this isolate may be pathogenic to sugar beet.

These findings prompted investigations to determine whether pigweed is susceptible to one or more physiologic races of R. solani, and whether this fungus can persist in soil in association with the infected tissues of this weed undergoing decomposition.

MATERIALS AND METHODS

The two fields of sugar beet and the field of alfalfa selected had been cropped previously to sugar beet, field bean and sugar beet, respectively. About 600 pigweeds were collected at random from each of the three fields during the last week of July 1958. At this time, the sugar beet field previously cropped to sugar beet had a high incidence of crown rot incited by R. solani. A moderate amount of crown rot was detected in the second sugar beet field previously planted to beans. The alfalfa had a low incidence of root and stem rot.

The number of pigweeds with diseased roots and/or stems was recorded and, subsequently, isolations were made from 100 infected plants from each field. Small pieces of tissue were excised from diseased roots and stems and thoroughly washed in tap water. Six isolations were made from as many roots and four from lesions on the stem of each plant. The washed tissues were then transferred to sterilized filter paper and dried at 25°C for 3 hours before transferring to Petri plates containing water agar (2%, pH 5.5). An incubation of 48 hours at 25° was sufficient for the establishment of R. solani. Cultures of R. solani were maintained on slants of potato-dextrose agar at 13°C.

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Physiologic races of R. solani were differentiated on the basis of gross morphologic characteristics of the colonies grown on various synthetic media and/or on the basis of pathogenicity on different hosts. Isolates of R. solani exhibiting different morphological characteristics when grown on PDA were transferred to Petri plates containing one of five media and incubated 18 days at 25°C. The five media employed were PDA, malt agar, Czapek Dox agar, V-8 juice agar and lima-bean agar. Differentiation of physiologic races on these media was based on the color and morphology of the colonies and sclerotia.

Pathogenicity tests of the isolates of R. solani were made during the winter of 1958 in the greenhouse at 25°C. Inoculum for infesting sterilized field soil from irrigated fields of western Nebraska was increased in soil supplemented with corn meal as described by Boosalis (1). After the appropriate inoculum was thoroughly mixed with soil in each 5-inch pot, the infested soil was incubated 3 days before planting. For the control treatment, the sterilized soil was supplemented with soil-corn meal medium. About 15 disinfected seeds were planted in each pot for the pathogenicity test with bean and alfalfa. The varieties of field bean and alfalfa used were Great Northern No. 59 and Ranger, respectively. Readings on damping-off and root and stem rot were taken 20 days after planting. Because of the low and uneven germination of seeds of pigweed, about 50 seeds were planted in each pot. Final readings on root and stem rot were taken about 25 days after planting. Each isolate was replicated four times for each of the crops and pigweed tested. The pathogenicity test was repeated with concordant results.

The pathogenicity of the isolates of R. solani was also tested on the potato variety Progress grown in the greenhouse and on the sugar beet variety Great Western 359R in the field. The toothpick method described by Schuster, et al. (3) was used for inoculating sugar beet. A toothpick infested with the appropriate isolate of R. solani was inserted into the base of 3-week-old stems of potato and incubated for 2 days in a humidity chamber at 25°C. These plants were then transferred to a greenhouse at 25° and incubated for 10 days before taking readings. At least four stems were inoculated for each of the isolates tested. Appropriate control treatments were also included.

Sugar beets were inoculated in the field on July 14, 1959. At least five beets were inoculated with the isolate tested. Readings on crown rot were taken on September 1, 1959.

Diseased roots and stems of pigweeds collected from fields of sugar beet and alfalfa in July 1958 were used to determine whether R. solani persists in soil in association with the decomposing, infected tissues. The diseased organs of pigweeds from each of the cropped fields were kept separate during the course of the experiment. The infected roots and stems from five pigweeds were buried 2 inches deep in each 5- inch pot filled with soil from the field from which the plants were collected. Each treatment was replicated four times. The pots were buried in a field near Lincoln, Nebraska in July of 1958. After 1 year, the buried roots and stems of pigweed were removed from the soil and isolations were made from these tissues. The method for isolating R. solani from decomposing, infected tissues consisted of washing the material in running tap water 4 hours, then submerging small excised pieces of tissue in 1% solution of sodium hypochlorite for 20 seconds, drying the pieces of tissue on sterile filter paper for 2 hours, and then transferring them to water agar (2%, pH 5.5). The surface disinfected tissues were incubated 48 hours at 25°C.

EXPERIMENTAL RESULTS

Sugar Beet Field with a High Incidence of Crown Rot: The highest amount of root and stem rot of pigweed occurred in the sugar beet field with the high incidence of crown rot. Dark brown lesions, some girdling the stems and roots, were noted on about 40% of the pigweeds. Based on the gross morphologic characteristics of the colony and on the pathogenicity tests, six physiologic races of R. solani were differentiated from diseased roots and stems of pigweed (Table 1).

Seventy % of the diseased pigweeds yielded only one physiologic race of R. solani. Furthermore, the majority of the isolates (60%) from different pigweeds were similar to the physiologic race of R. solani inciting crown rot of sugar beet. This particular race, designated as race 1, was moderately pathogenic in causing damping-off of alfalfa and nonpathogenic to potato and bean. Race 1 also caused stem and root rot of pigweeds. Race 2 was isolated from about 25% of the pigweeds yielding one race of R. solani. This race was innocuous on sugar beet, bean and alfalfa. However, potato and pigweed were highly susceptible to race 2 (Table 1). Race 3 was recovered from infected roots and/or stems of the remaining weeds (15%) infected with a single race of R. solani. The pathogenicity of race 3 was confined to pigweed and bean, causing severe root and stem rot of the latter.

Table 1. Pathogenicity of six physiologic races of Rhizoctonia solani isolated from diseased roots and/or stems of pigweed.

Race	Susceptibility of five kinds of plants				
	Sugar beet	Alfalfa	Bean	Potato	Pigweed
1	+	+	-	-	+
2	-	-	-	+	+
3	-	-	+	-	+
4	+	-	-	-	+
5	-	-	-	-	+
6	-	+	+	-	+
Control	-	-	-	-	-

Various combinations of races 1, 2 and 3 were also isolated from 32% of the pigweeds infected with two races of R. solani. In addition to these 3 races, however, race 4 was recovered from diseased roots and stems of a few of the weeds. Only sugar beet and pigweed were susceptible to race 4 (Table 1).

About 5% of the pigweeds were parasitized by three races of R. solani. Race 5 was isolated from the roots and stems of some of these pigweeds as well as races 1-4. Only pigweed was susceptible to race 5 (Table 1).

Individual plants of the remaining 3% of the diseased pigweeds from this sugar beet field yielded four races of R. solani. Various combinations of races 1, 2, 3, 4, 5 as well as race 6, were isolated from this group of pigweeds. Race 6 was pathogenic to alfalfa, bean and pigweed (Table 1).

These races of R. solani also differed in the gross morphological characteristics of their colonies and sclerotia when grown on the five synthetic media.

Diseased roots and stems of pigweed buried in soil for 1 year yielded races 1, 2, 3 and 4, with race 1 predominant. The relatively small number of pigweeds parasitized by races 5 and 6 may explain the failure to isolate these races from diseased tissues buried in soil.

Sugar Beet Field with a Moderate Incidence of Crown Rot: About 20% of the pigweeds from the sugar beet field with a moderate amount of crown rot were infected by R. solani. Nearly 80% of the diseased weeds yielded one race of R. solani. However, not all of these plants were infected with the same race.

Two races of R. solani were isolated from about 15% of the pigweeds, and three physiologic races were obtained from the remaining 5% of the diseased plants. Again not all of these plants were infected with the same combination of races.

Seven physiologic races of R. solani were recovered from the roots and/or stems of the 100 diseased pigweeds. Besides the six races previously described (Table 1), still another race, race 7, was isolated from the stem of one pigweed. Although race 7 could not be differentiated from race 5 on the basis of pathogenicity, it differed from race 5 in gross morphological characteristics. If more kinds of hosts had been employed, races 7 and 5, undoubtedly, may have been differentiated.

Race 4 was most frequently isolated from diseased pigweeds. About 60% of the weeds were infected with this race. Race 1 was found in about 20% of pigweeds, whereas races 3, 4, 5 and 6 were obtained from 10% of the plants.

Isolations from roots and stems buried in soil for 1 year yielded races 2, 1, 5 and 3, in that order of prevalence. Another isolate of R. solani, morphologically unlike the previous seven races, was isolated from the buried tissues nearly as frequently as race 1. It is conceivable that this new race, designated as race 8, colonized the tissues after they were buried in soil.

Alfalfa Field with Low Incidence of Root and Stem Rot: Although alfalfa was not adversely affected by R. solani, there was a high incidence of root and stem rot of pigweeds in this field. About 30% of the weeds were infected with R. solani. Moreover, this fungus had caused damping-off of many pigweeds. To the authors' knowledge this is the first report of damping-off of pigweeds incited by R. solani in the field.

Isolations from the diseased pigweeds yielded 10 physiologic races of R. solani. Races 9, 10, 11, 12, and 13 were isolated from pigweeds in addition to races 1, 2, 3, 5, and 6. The host range employed in this study was inadequate to differentiate races 9-13 from race 5. Consequently, the five new races, 9-13, were separated solely on the basis of their gross morphological characters.

Races 1 and 6, pathogenic to alfalfa, comprised about 60% of the isolates from the diseased weeds. About 20% of the pigweeds were infected with race 3. Less than 10% of the plants contained races 2, 4, 5, 8, and 9, and races 10 and 11 were found in each of two plants.

Again the majority of diseased pigweeds, 80%, were infected with a single race of R. solani. With the exception of races 4 and 9, all races just listed were isolated from these plants. Races 2 and 3 were obtained from individual weeds from 20% and 6% of the plants, respectively. The most commonly isolated races from single plants yielding two races were 1, 6, 3 and 5. One pigweed was infected with 4 races -- races 1, 6, 3 and 4.

Races 1, 2, 4, 6, 8 and 10 were recovered from tissues of pigweed buried in soil 1 year. Furthermore, two new races, 14 and 15, were also isolated from these tissues undergoing decomposition. These two races were differentiated from race 5 by their morphological characteristics. It was interesting to note that race 6 comprised about 55% of the isolates from pigweeds buried in soil.

DISCUSSION

Results from this study indicate that pigweed plays an important part in the epidemiology and ecology of Rhizoctonia solani inciting root and stem rot of irrigated crops in western Nebraska. The susceptibility of this weed to at least 15 physiologic races of R. solani, coupled with the ubiquitous nature of this host, explains in part how this fungus persists for protracted periods in soil in the absence of a susceptible crop. It was apparent from this investigation that not only do pigweeds sustain growth of many races of R. solani, but the infected pigweed tissues that are undergoing decomposition in soil harbor viable inoculum of the fungus for at least 1 year. This does not imply, however, that the plant residue from pigweeds infected with R. solani is the only source of inoculum of the different races. In this connection, Boosalis and Scharen (2) presented evidence indicating that R. solani, pathogenic to sugar beet, retains its viability in association with plant debris particles of sugar beet for at least 7 months. Results from other studies, not reported here, also indicated that several races of this fungus may persist in soil in association with plant residue from infected lamb's quarters, Chenopodium album.

Results from preliminary investigations in the greenhouse also indicate that R. solani may be disseminated passively for short distances by irrigation water and/or by wind. Whether this plant residue-inhabiting fungus can be transported for long distances by these two agents and still retain its viability is a question for further study.

In the light of the results from this study it seems advisable to eradicate pigweeds from irrigated fields to minimize losses from root and stem rot incited by R. solani. The common occurrence of this weed in sugar beet fields undoubtedly is an important factor contributing to high incidence of crown rot in many of the irrigated fields.

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PHYTOPHTHORA CANKER OF MACADAMIA TREES IN CALIFORNIA

George A. Zentmyer

In recent years increasing plantings have been made of macadamia nut in southern California, although the largest acreages of this tree occur in the Hawaiian Islands and in its native habitat in Australia (1). To date this plant has been singularly free of diseases.

In October 1959 trunk cankers were observed on two macadamia trees in a commercial planting in Vista, California. The trees had been replanted in an area where avocado trees had been removed because of Phytophthora root rot, caused by *Phytophthora cinnamomi*. One of these trees was a *Macadamia integrifolia* seedling, the other was clone 508 of *M. integrifolia* grafted on a *M. tetraphylla* seedling. In the latter case the canker occurred on both rootstock and scion.

One canker had progressed to the extent of girdling the base of a branch approximately 3/4 inch in diameter. Excision of the bark revealed brown discoloration in bark and outer wood, extending in a narrow streak from ground level to the girdled lateral branch 18 inches above the ground. No external symptoms of the canker were visible on the bark. The second tree had two slightly sunken and rough areas on the lower trunk; discoloration in bark and outer wood was also found here.

Culturing pieces of bark and wood tissue from the active-appearing margins of the lesions on corn meal agar resulted in isolation of *Phytophthora cinnamomi* from both trees.

Sixteen macadamia seedlings (8 *M. tetraphylla* and 8 *M. integrifolia* seedlings), provided by Dr. W. B. Storey, Department of Horticulture, University of California, Riverside, were inoculated in October 1959 with the isolate of *P. cinnamomi* obtained from the *M. integrifolia* seedling (isolate SD 808), and with an isolate from avocado roots (isolate SB 216). The seedlings were vigorous and ranged from 10 to 14 inches in height. Slits were made in the bark of the lower stem of the seedlings, 5-mm disks from a potato-dextrose agar culture of the fungus were placed in the wound, and the wound was wrapped with adhesive tape. Four other trees were not wounded but inoculum was placed on the stems, covered with moist cotton, and wrapped with adhesive tape. Four trees were wounded but not inoculated.

The first visible symptoms from the inoculations appeared in 3 weeks, and by 4 weeks after inoculation 9 of the 12 seedlings which were wound-inoculated had developed cankers of varying extent. *P. cinnamomi* was readily re-isolated from lesions on these stems. Two of the *M. integrifolia* and one of the *M. tetraphylla* seedlings died within 4 months after inoculation. Some gum exudations and sunken, cracked areas on the stems were apparent on other seedlings. No lesions developed from inoculations made without wounding. Both isolates of *P. cinnamomi* were pathogenic to macadamia. No additional seedlings developed cankers up to 10 months after inoculation.

These results indicate that trunks of branches of macadamia trees are susceptible to *P. cinnamomi* when the fungus is introduced through a wound. Roots of the macadamia are evidently quite resistant to *P. cinnamomi* as indicated by consistent lack of root rot development when seedlings of this tree have been planted in soil heavily infested with *P. cinnamomi* (2).

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STORAGE DECAYS OF DOMESTICALLY GROWN CHESTNUTS

W. R. Wright[1]

Abstract

The advent of domestic chestnuts on the market focuses attention on the market potential of this product. In a study of storage decays of Chinese chestnuts made in cooperation with the U. S. Horticultural Field Station, Meridian, Mississippi, fungi representing 12 genera in addition to various bacteria and yeasts were recovered from kernel decays. Phoma castanea Peck, Dothiorella sp., Cytodiplospora castaneae Oud., and Pestalotia spp. accounted for over 75%.

Phoma, Dothiorella, and Cytodiplospora were not recovered from infected nuts exposed to 283, 000 rads of gamma irradiation. Phoma, Cytodiplospora, and Pestalotia cultures were killed at 243, 000 rads. Dothiorella cultures survived a dosage of 250, 000 rads.

High decay incidence encountered suggests that bulk storage of chestnuts is unsatisfactory. Shelling to permit careful kernel grading is considered to be of prime importance. Merchandising the kernels would then require proper packaging and quick freezing for year-around consumption. This is being tried on a commercial basis.

INTRODUCTION

In 1958 Georgia-grown Chinese chestnuts (Castanea mollissima) were first sold commercially as food nuts. The initial offering was increased to 40, 000 to 50, 000 pounds in 1959, and prospects are for continued expansion.

The successful establishment of blight-resistant chestnut stands in the United States would provide a valuable cash crop for growers. At the present time domestic chestnuts face stiff competition from imports which have dominated the market for many years. Chestnut imports for 1958 were 14, 975, 538 pounds with a dollar value of 1, 486, 705. Preliminary estimates for 1959 were 19, 066, 082 pounds with a dollar value of 1, 837, 967[2].

In order to compete, the domestic product must be of first quality. Domestic-grown chestnuts are subject to decay, the amount and types varying with the seasonal, varietal, and geographical factors. This paper is the result of a cooperative study of the storage decays of chestnuts from established stands at the U. S. Horticultural Field Station, Meridian, Mississippi.

MATERIALS AND METHODS

Specimens of orchard material sent at intervals throughout the growing season, and harvested nuts stored at Meridian, Mississippi, or the Chicago Market Pathology Laboratory formed the basis for the observations recorded.

The shell and integument were removed from 1855 nuts in order to accurately determine initial infections. The location, number, and physical characteristics of the lesions on the kernels were recorded. Over 3000 potato-dextrose agar cultures were made of infected tissues and of integument fragments from both crown and calyx areas.

The irradiation experiments conducted utilized the high-level gamma irradiation facilities at the Argonne National Atomic Source, Lemont, Illinois. In one experiment six No. 2 cans containing 25 chestnuts each were exposed to 56, 000, 87, 628, 160, 652, 283, 880, 419, 888 and 867, 160 rads. The irradiated nuts and controls were examined and PDA cultures made of diseased or suspect tissue.

In a subsequent experiment a replicated series of three PDA tube cultures of the four dominant fungi encountered in this investigation were exposed to complementary short and fast flux rates of 85, 100, 92, 200, 126, 000, 131, 000, 144, 000, 150, 000, 172, 000, 187, 000, 204, 000, 210, 000, 243, 000 and 250, 000 rad doses to test the radiation-sensitivity of the organisms.

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[1]Pathologist, Market Quality Research Division, Agricultural Marketing Service, United States Department of Agriculture, Chicago, Illinois. The author wishes to gratefully acknowledge the assistance of Mr. A. C. Gossard and Mr. L. J. Kushman for supplying the plant material and making valuable suggestions during the course of the study; to Marie L. (Farr) Lack for her identification of Macrophoma sp., Phoma castanea, and Pestalotia quercina; and to Drs. Marvin E. Fowler and J. D. Diller for their efforts in obtaining orchard material infected by Cytodiplospora.

[2]Estimate furnished by the Foregin Agricultural Service from correspondence with Mr. C. M. Purvis, Director of Statistics.

Following irradiation, transfers of all treated material and controls were made to fresh PDA tubes for observation.

A 4-plate, replicated series of temperature tests was made to determine the temperature tolerances of the key fungi involved in this study. The inoculum was given 24 hours at 72° F to establish itself on the potato-dextrose agar before being placed at the desired temperature for 1 week.

RESULTS

Visible infections were observed on 62% of the kernels. Analysis of infection patterns on the kernels showed that in 43% the entire fruit was involved, or individual lesions were so universally distributed that the point of initial infection could not be accurately determined. Thirty-four% showed infections starting at the crown; 14% at the side; 10% at, or near, the calyx.

A fungus identified as Phoma castanea Peck accounted for 34% of the fungi recovered from kernel lesions, and 27% of the fungi obtained from calyx and crown integument plantings. This fungus was encountered on catkins, leaves, and twigs; often permeating the soft crown tissues of preharvest burrs. An infection court for inoculation of the kernel was thus provided.

Initial infection is first evident as tiny brown to black specks. These infections enlarge, or coalesce, eventually involving the entire kernel. Affected tissues are chalky to brown in color, and punky to friable in texture. Mycelium, when present, consists of a light, white weft scarcely concealing the lesion. Fruiting is favored by moderately high humidity, tends to be vigorous, and covers the affected area with fragile, sometimes pubescent, fruiting bodies exuding copious light tan spore masses (Fig. 1A).

Appearance in culture varies somewhat, but mycelial production is usually slight, zonate, and white to very light gray. Fruiting occurs readily and is marked by the appearance of small individual, or closely aggregate, fragile pycnidia 30-250 μ in diameter. The brown to dark brown bases are soon surmounted by a light tan spore mass. Spores range from 2.13 to 5.2 x 5.7 to 10.4 μ, averaging 3.57 x 7.0 μ. They are short-elliptic to ovoid, sometimes tapering sharply at one end, and usually display a guttule at one or both ends. They are continuous, hyaline, and may or may not be granular in content (Fig. 3E). The mycelium is septate, often extremely transparent outlined by a highly refractive wall, septa, and inclusions. Anastomosing is common. An ostiole has not been observed in pycnidia on culture media, or on nut kernels (Fig. 2B). The bodies appear to rupture irregularly under pressure from the underlying spore mass. Spores are borne on simple conidiophores 5-15 μ long. The organism tends to be strongly intercellular on the host. Minimum temperature for growth of this fungus occurred at 35° F, optimum at 80°, and a maximum at about 97°.

A Dothiorella sp. accounting for 27% of the fungi recovered from decayed kernels and 21% of the fungi obtained from calyx and crown integument plantings ranked next in importance.

This decay also begins as small dark brown to black, individual, or clustered lesions. In a more advanced stage lesions are burnt brown to black, shading to a water-soaked brown at the border. Internal characteristics of the decay show a water-soaked browning of the tissues with progressive drying and darkening as the fungus matures. Eventually this results in the entire kernel becoming punky to friable, and gray to dark gray. Surface lesions are usually covered with gray mycelium which, in severe decay, compacts into stromatic bodies supporting pycnidia (Fig. 1C). These range in size from 135 to 350 μ long by 100 to 250 μ wide (Fig. 2A). Initial growth on PDA is white, but colonies soon assume a gray cast spreading from the center of the plate. Mycelium in mature cultures is uniformly dark gray and moderately flocculent. Pycnidial formation on PDA is infrequent. Spores are hyaline, ellipsoid, and range in size from 16.67 to 30.9 x 4.76 to 8.33 μ, averaging 22.9 x 5.36 μ (Fig. 3B). Spores are predominantly continuous; in rare instances 1 or 2 septate spores have been observed. Conjugating spores were occasionally noted. Minimum temperature for growth of this fungus is about 35° F, optimum at about 80°, and maximum at about 95°.

A fungus identified as Cytodiplospora castaneae Oud. accounted for 9% of the fungi recovered from kernel lesions, and 9% of the fungi recovered from integument plantings. This fungus, in association with its perfect stage Cryptodiaporthe castanea (Tul.) Wehm. was noted by Fowler (4) as a cause of cankers or dieback on trunks, limbs, or twigs of Asiatic chestnuts in the Eastern United States. On PDA plates mycelium is appressed, concentric, and dark. Pycnidia are small, scattered, and mostly individual on twig, leaf, and culture media. They range from 50 to 150 μ in culture but are somewhat larger on kernel tissue where they tend to be closely aggregate. Fruiting occurs freely both on the surface, and within the kernel tissue

FIGURE 1. Chinese chestnut kernels infected by A -- Phoma castanea. B -- Pestalotia sp. C -- Dothiorella sp.

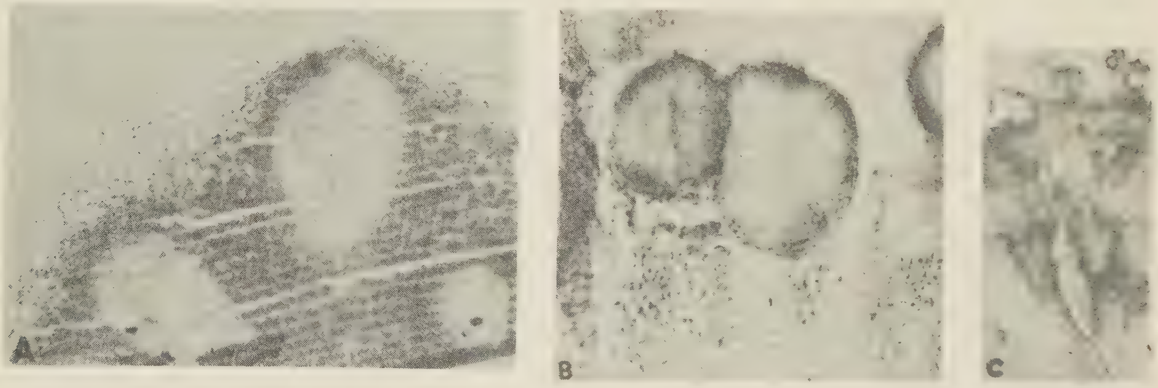

FIGURE 2. Fruiting on Chinese chestnut kernels by A -- Dothiorella sp. 160X. B -- Phoma castanea 240X. C -- Cytodiplospora castaneae 50X.
[Magnifications for all figures reduced by approximately 25%]

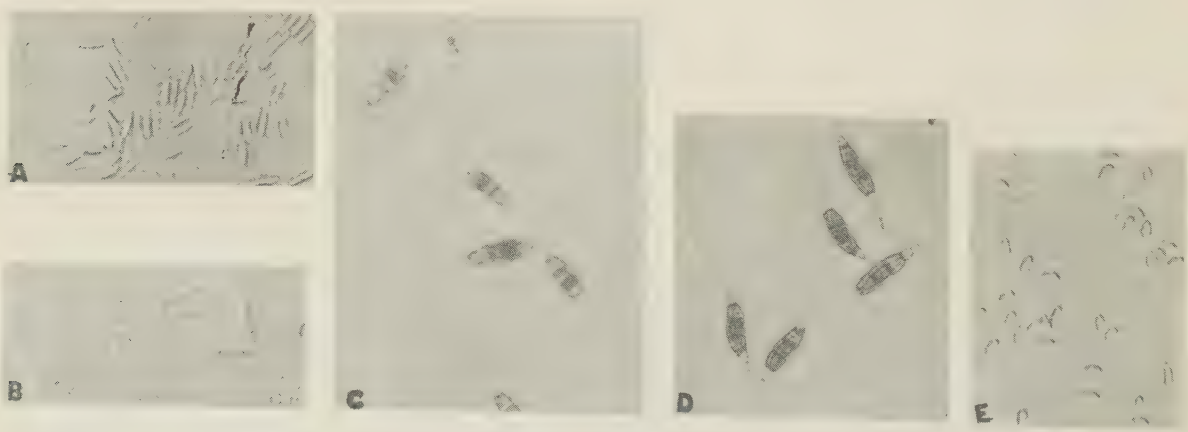

FIGURE 3. Spores of A -- Cytodiplospora castaneae. B -- Dothiorella sp. C -- Pestalotia sp. D -- Pestalotia quercina. E -- Phoma castanea 540X.

(Fig. 2C). An ostiole has not been noted in culture, but has been observed in the fungus growing on stems, leaves, and kernels. Colorless to muddy-gray spore droplets surmount mature pycnidia. Spores are mostly elliptical, hyaline, straight or slightly curved, and usually display a faint central septum or pseudoseptum (Fig. 3A). Occasionally they are more or less dumbbell shaped and heavily granular. Size varies from 2.6 to 5.2 x 6.2 to 11.4 μ, averaging 4.4 x 9.3 μ. They are borne on short simple sporophores. Minimum temperature for growth of this fungus is about 32° F, optimum 80° to 85°, and maximum about 95°.

Two species of Pestalotia were recovered from 7% of the kernel decays, and 18% of the calyx and integument plantings. Growth characteristics on PDA tubes were similar, presenting a pure white, felty appearance with scattered jet black spore masses exuding from underlying pseudopycnidial bodies.

One isolate yielded 5-celled spores; the end cells hyaline, the central cells equally colored or the upper two slightly darker. At the apex were 2-4 (3) widely divergent setae 6.0-16.0 (10.4) μ long. The conic basal cell supported a short, straight pedicel 2-5.25 (4.6) μ long. Spore length was 20.8-26.0 (22.5) μ. The olivaceous median cells measured 14.5-18.2 x 5.2-7.8 (15.75 x 5.7) μ.

Spores were erect, or slightly curved, hardly constricted at the septa (Fig. 3D). This Pestalotia appears identical with type material of Pestalotia quercina Guba.

Spores of the second isolate were likewise 5-celled, the end cells hyaline. The upper median colored cells were usually slightly darker than the lower, and usually exhibiting a dark band between the upper cells. At the apex were 2-4 (3), divergent, rarely branched setae 20.8-36.4 (33.18) μ long. The conic basal cell supported a straight, slanted, or curved pedicel 5.2-15.6 (10.5) μ in length. Spores were 20.8-26.0 (24.7) μ long. The colored cells measured 13.0-18.2 x 5.2-7.2 (15.75 x 6.3) μ (Fig. 3C).

Both species have been recovered from initial and advanced tissue decay but records are not complete enough to determine their relative status. Lesions were tan, slightly sunken, and often delineated by a dark border. Scattered fruiting pustules may be present (Fig. 1B). Affected tissue is chalky, or very light gray in color, and is punky to friable in texture.

Temperature reactions are similar for both species with a minimum of 40° F, and optimum of 75° to 80°, and a maximum of about 95°.

In descending order of importance with respect to percentage recovery of other fungi, bacteria, and yeasts from kernel infections, and crown and calyx integument plantings respectively were species of Phomopsis (5 and 4.7); Penicillium (3.99 and 12.42); Alternaria (1.82 and 2.16); Papulospora (1.69 and 6.8); Macrophoma (1.45 and 5.21); Bacteria (1.33 and 3.91); Fusarium (1.09 and 3.18); Yeasts (.97 and 4.14); Rhizopus (.60 and 2.78); Cladosporium (.12 and 3.62); undetermined were 3.14 and 3.24%.

In addition to fungus decays, nuts were observed in which white starch areas occurred just beneath the surface. These usually shallow areas varied greatly in size, shape, and number and were not confined to any particular portion of the kernel. It was apparently the same condition noted by Fowler and Berry (5). Approximately 1000 kernels were checked to determine the prevalence of this condition. Seventeen % were affected. Seventy % of the cultures attempted were sterile. This condition is considered to be physiological in nature; the miscellaneous fungi recovered were apparently present in a secondary capacity. There was no evidence that insects might be a contributing factor.

Phoma, Dothiorella, and Cytodiplospora were isolated from infections on nuts exposed to irradiation doses up to, and including, 160,000 rads. None were similarly recovered from nuts exposed to 283,000 rads, or over. Kernel discoloration was objectionable at 419,888 rads and beyond. Pestalotia was not a factor in this test.

Phoma, Cytodiplospora, and Pestalotia grew well following transfer to fresh potato-dextrose agar after exposure of PDA cultures to 204,000 rads. Similar transfers of tubes which had received 243,000 rads failed to grow. Dothiorella transfers from cultures exposed to the test maximum of 250,000 rads grew well.

DISCUSSION

Examination of arboreal material sent in to the Chicago laboratory indicated that the fungi most commonly associated with kernel decay were present the year around as saprophytes, or weak parasites on dead or dying twigs. This provides a source of inoculum for leaf, floral, and fruiting parts in turn.

Microscopic examinations, observations of material in damp chambers, and cultures attempted throughout the growing season, plus infection loci on nuts examined, have suggested

the burr crown to be a principal avenue of attack for the storage organisms under consideration. Infection potential may be established early, fungus elements having been noted within the shells of immature nuts examined in June. Despite such occasional encounters the nature of the fungi recovered suggests that progress toward kernel decay is largely conditioned by the aging of the protective husk, and shell tissue. Thirty-four % of the kernel infections recorded during this study were initiated at the upper half of the nut, largely concentrated at the crown. This portion corresponds to the point of connection of nut integument to the nut shell, and of nut shell to the burr. These provide the vascular supply route for the nourishment of the developing kernel. This area remains relatively soft and penetrable until the nuts are mature. Hyphal elements have been noted in abundance in the fleshy median crown burr tissues, nut crown, and attached integument, often overlying initial infections on preharvest nuts. The inherent weakness of the crown area of chestnuts has been noted by Riccardo (9) who immersed chestnuts in an aqueous solution of carmine dye to demonstrate that the dye penetrated in a manner similar to that of the microorganisms. Other investigators have found the calyx area to be most susceptible to decay (1, 3, 5, 6, and 7); the microorganisms apparently gain entrance through the withered remains of the styles, or through a crack in the pericarp at the apex of the nut. It is probable that either mode of attack may predominate during certain seasons, and with differing types of pathogens.

The pattern observed in this study indicates that, of those nuts destined to decay in storage, most harbor the decay organisms within the shell prior to harvest, and that many of the kernels are initially infected at harvest or following a relatively short storage period by the pre-established fungus elements within the pericarp. This contention is supported by the lack of consistent correlation between the amount of decay present and the length of time in storage; by the relationship between the fungi recovered from the preharvest and the postharvest chestnut material examined; and from microscopic and macroscopic observation of chestnut material throughout the season. That the kernel-decay potential is present within the pericarp is illustrated by one experiment wherein plantings were made from crown and calyx integument fragments from 340 uninfected storage nuts. Fifty-three % of the plantings were successful, with 78% consisting of the four fungi dominating the isolates from kernel decays. Preharvest infection has been observed, or suspected, by a number of investigators (2, 5, 6, 7, and 8).

The rather high percentage of decay encountered in harvested chestnuts from the Meridian Station, while not necessarily typical either from the standpoint of incidence or microflora involved, does emphasize the importance of quality control by distributors of this product. Such control is difficult, since there is usually no visible indication of decay. It has become increasingly apparent throughout the course of this study that only exposure of the kernel to permit careful inspection can guarantee a superior product. In this connection one Southern distributor of U. S. grown chestnuts claims to have devised a method of shelling which will permit such inspection. The feasibility of marketing the kernels in polyethylene bags, and as a frozen product for year-around consumption is also being explored. This latter development is especially significant since it may provide a means of salvaging those nuts which show only initial infections at harvest. These would not be commercially objectionable if frozen immediately, but would represent a loss under prolonged bulk storage. This investigation suggests that such marketing practices should be encouraged for the presentation of a quality product, a vital requirement for an infant industry.

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DODINE EFFECTIVE AGAINST BACTERIAL SPOT OF PEACH

Robert H. Daines

Information on the control of bacterial spot of peach (*Xanthomonas pruni* (E. F. Sm.) Dows.) by chemical means has accumulated slowly despite the widespread and destructive occurrence of the disease. Many synthetic fungicides have been used as sprays or dusts in field trials, but possibly only captan (N-trichloromethylmercapto-4-cyclohexene-1, 2-dicarboximide) at 4 pounds per 100 gallons has approached zinc sulfate-lime (1-4) in effectiveness. Zinc sulfate-lime has become the standard fungicide against this disease since Roberts and Pierce (4) first reported its use in 1929, despite the fact that it affords only about a 50% reduction of spot on the fruit and is less effective in preventing infections on the foliage. Daines (1) suggests that this poor performance of surface protectants is probably due to the method by which ingress of the pathogen occurs. The bacteria that cause infections of the peach swim into or are carried into the stomatal or lenticel chambers in the water entering these chambers. Ingress may therefore occur in minutes or even seconds, in contrast to the hours taken when ingress results from a growth process. Chemicals deposited on the surface, therefore, have little opportunity to kill all the bacteria splashed onto a leaf during a rain before ingress occurs. Data presented by Daines and Gray (2) suggest that control of this disease with streptomycin is improved by increasing the adsorption of the antibiotic. Perhaps a limited systemic activity is required for a successful bacterial spot bactericide.

In an experiment on rusty spot control, Goldeneast peaches sprayed with dodine (*n*-dodecylguanidine acetate) at 2 pounds per 100 gallons ripened early, showed an increased yellow ground color and, when green, exhibited darker green spots over the peach. This irregular green color suggested adsorption of the dodine. Accordingly, in the 1960 field trials dodine was included in a bacterial spot test at three concentrations, at 1/4-, 1/2-, and 1-pound concentrations, alone and with 1-pound of captan. These treatments were compared with zinc sulfate-lime, captan at 2, 3 and 4 pounds, and sulfur on eight randomized trees per treatment. All treatments, beginning with the shuck split and continuing until harvest, were applied on a 10-day schedule using an eight cluster nozzle gun and 300 pounds' pressure. The comparative effectiveness of these materials in controlling bacterial spot is shown in Table 1.

Table 1. Incidence of bacterial spot on peach fruits sprayed with various materials.

Treatment and concentration (in pounds)[a, c]	Total fruits affected: angle[b]	Total fruits affected: %	Fruits severely affected: angle	Fruits severely affected: %
Sulfur 4	44.12	48.6	27.08	20.9
Captan 2	45.81	55.2	28.80	23.7
Captan 3	45.02	51.7	29.55	24.7
Captan 4	39.01*	42.6	23.86*	17.0
Zinc sulfate-lime 5.5 - 12	32.31**	29.0	16.60**	8.6
Dodine 1/4	37.65**	39.4	21.92*	14.5
Dodine 1/2	28.37**	24.0	13.16**	5.4
Dodine 1	17.65**	9.4	6.98**	1.7
Dodine 1/4, captan 1	37.13**	37.1	21.45**	13.9
Dodine 1/2, captan 1	29.32**	24.4	16.42**	8.2
Dodine 1, captan 1	25.31**	20.4	10.94**	4.2
L.S.D. .05	4.8		3.97	
.01	6.3		5.26	

[a]Sprays applied on a 10-day to 2-week schedule beginning with the shuck split.
[b]Angle = arc sin $\sqrt{\text{percentage}}$.
[c]Parathion was used in all treatments at the shuck split, 1st, 2nd and 5th cover sprays.

In this experiment with the sulfur treatment serving as check, captan at 4 pounds provided protection that was significant at the 5% level, whereas zinc sulfate and lime and all dodine treatments showed control that was significant at the 1% level. This was true for both the total and severely infected fruits. The most significant thing about this experiment was the performance of dodine at the 1-pound level. At this concentration the dodine treatment provided highly significant control over zinc sulfate-lime. So far as the writer is aware, this is the first time

in New Jersey that a fungicide has given significantly better protection against bacterial spot than zinc sulfate-lime. These data suggest that captan at 1 pound did not alter the efficiency of dodine at the 1/4- or 1/2-pound rates. However, where total bacterial spot is considered, the control provided by the 1-pound dodine treatment was highly significant over the captan 1-pound dodine 1-pound treatment. The data presented for severe spot infections indicate that dodine at 1 pound just missed showing a significant improvement over the captan 1-pound dodine 1-pound mixture.

At no time during the course of this experiment did the foliage of the Sunhigh peaches sprayed with dodine show symptoms of injury, but the green spotting already referred to was observed on peach fruits sprayed with dodine at the 1-pound level. Although this spotting was observed on fruits sprayed with the 1/2-pound dosage, it was rather hard to find on the trees in this treatment, and none was found on the 1/4-pound-treated trees. At harvest time, areas on some peach fruits sprayed with dodine at the 1-pound level showed a yellow color rather than red. These yellow areas were small, 1/4 inch in diameter, except when they occurred at the lower tip of the peach where they were observed up to 1 inch in diameter. Fruits showing this spotting averaged about 1% of the total fruits where dodine at 1 pound was used. The number of spotted fruits at harvest time was less on the captan 1-pound dodine 1-pound treatment than on the one receiving 1 pound of dodine alone.

While the spotting of the fruit was not serious in this test, more needs to be known about the effect of other chemicals on the efficiency of dodine in disease control as well as possible phytotoxic effects on the varieties that are susceptible to bacterial spot under various environmental conditions. Since this green and yellow spotting of fruit sprayed with dodine did occur, investigators using this material on peaches should watch closely for this or other symptoms of injury. The possibility of phytotoxicity is further stressed by the report of Diener and Carlton (3), in which they relate, without justification from the data presented, that dodine produced leaf injury to sprayed peach trees and that captan apparently had a safening effect on dodine.

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THE OCCURRENCE OF SPINACH RUST IN COLORADO[1]

Nagayoshi Oshima[2], W. J. Henderson[3], and L. E. Dickens[4]

In May 1959, specimens of spinach leaves heavily infected with aecia of spinach rust (*Puccinia aristidae* Tracy) were sent to the office of the Extension Plant Pathologist at Colorado State University from Adams and Pueblo counties in Colorado. Since desert saltgrass (*Distichlis stricta*) is the alternate host of this heteroecious long-cycle rust, the presence of uredial and telial stages on saltgrass in these counties was suspected.

Ramsey and Smith (3) in 1944 reported heavy rust infection on Colorado spinach from the river-bottom near Denver, and heavy uredial and telial infection on saltgrass near the spinach field was also reported.

Earlier, Pool and McKay (2) reported that *P. aristidae* caused moderate damage to sugar beets around Rocky Ford, Colorado in 1912-1913 and noted that the telial stage was abundant on saltgrass in the region.

In order to obtain information on the extent of uredial and telial infection of saltgrass by *P. aristidae* in Colorado, so that warning may be issued to the farmers before planting spinach, a survey was made throughout Colorado in the summer of 1959.

As shown on Figure 1, during the survey of 1959, infection of saltgrass by uredial and telial stages of *P. aristidae* was found in Larimer, Weld, Adams, Elbert, Lincoln, Cheyenne, Kiowa, Bent, Crowley, Otero, Huerfano, Pueblo, Alamosa and Delta counties. As shown here, spinach rust was present on saltgrass in not only eastern Colorado but also west of the Continental Divide. Especially heavy infection on saltgrass was found in Larimer, Weld, Adams, Crowley and Delta counties.

Since these rust-infected saltgrasses often occurred far away from fields of spinach or sugar beets, it appeared that *P. aristidae* completes its life cycle in the absence of cultivated susceptible hosts, by causing aecial infection on wild plants. Members of the following families are listed by Arthur (1) as the aecial host of *P. aristidae* in Colorado: Scheuchzeriaceae, Polygonaceae, Chenopodiaceae, Amaranthaceae, Nyctaginaceae, Aizoaceae, Caryophyllaceae, Fumariaceae, Cruciferae, Capparidaceae, Loasaceae, Polemoniaceae, Boraginaceae and Solanaceae. Among these families, numbers of species included are greatest in Chenopodiaceae and Cruciferae.

As the survey indicates, the infection of saltgrass by *P. aristidae* in Colorado is very extensive, and it seems able to survive for many years without the presence of susceptible spinach or sugar beet crops by causing aecial infection on wild plants and thus completing the life cycle. When spinach is planted near saltgrass colonies, heavy infection of spinach by *P. aristidae* is likely to occur. Therefore, it is advisable to destroy saltgrass colonies in the vicinity before planting spinach.

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[1] Published with the approval of the Director, Colorado Agricultural Experiment Station as Scientific Series Paper No. 682.

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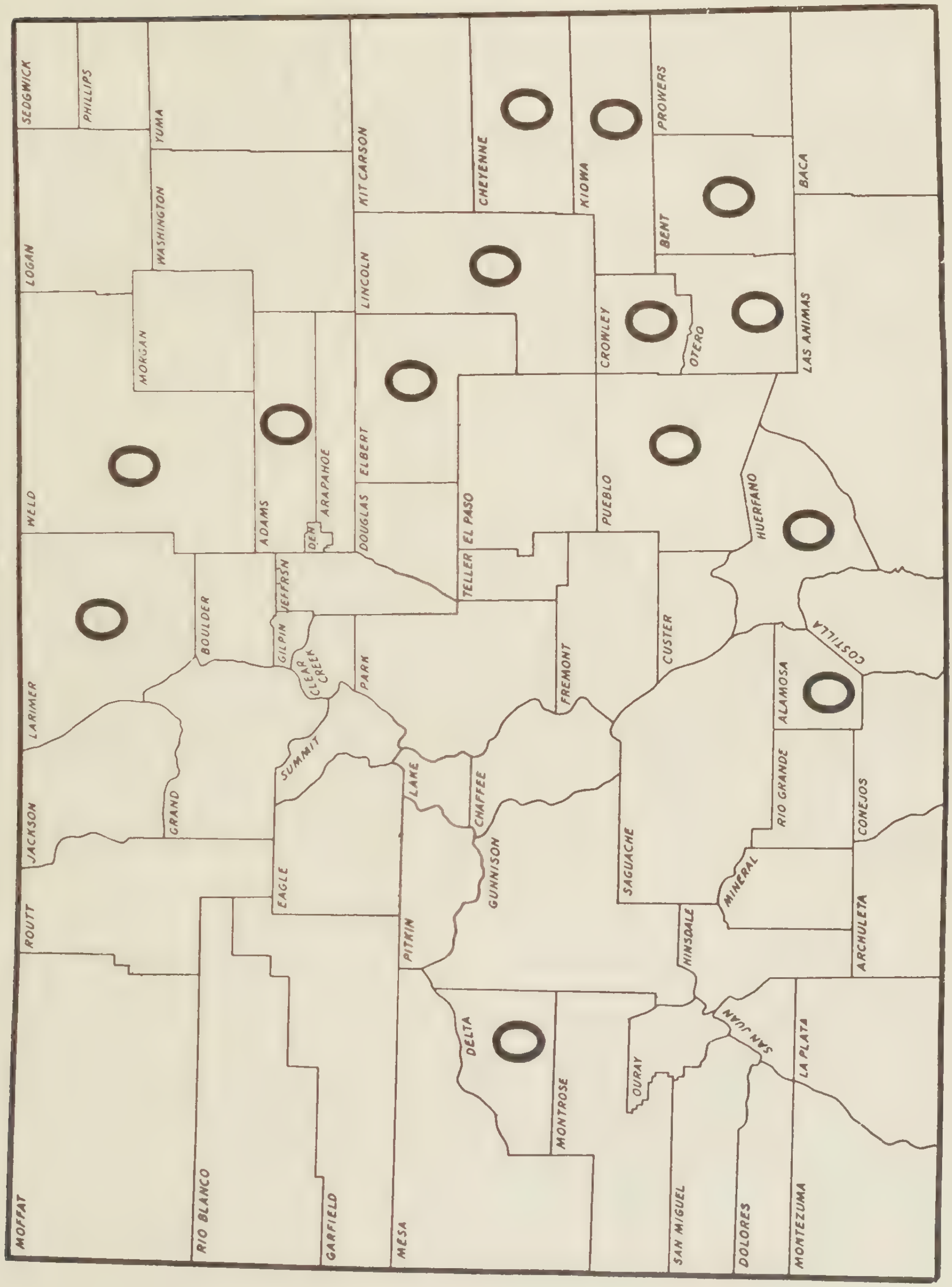

FIGURE 1. Distribution of *Puccinia aristidae* on saltgrass (*Distichlis stricta*) in Colorado in 1959.

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STUDIES ON METHODS OF APPLICATION OF EMULSIFIABLE DBCP AROUND LIVING CITRUS TREES

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Abstract

Different amounts of DBCP were applied around living citrus trees by overhead sprinklers, furrows and basins. The distribution of the chemical and control of the citrus nematode in a number of soils was determined. The distribution of DBCP in the row middle and drip of tree were similar; however, no DBCP was detected in any soil samples taken from under the tree when it was applied through overhead sprinklers. The application of the fumigant in furrows did not provide satisfactory distribution of DBCP throughout the treated area. Basin irrigation for the application of DBCP in water produced the most uniform distribution of the fumigant and control of the citrus nematode. Distribution of the DBCP was obtained to greater depths by applying larger amounts of water than ordinarily were used during a regular irrigation in the test groves.

INTRODUCTION

Arizona and California citrus growers have shown a great deal of interest in the use of 1,2,dibromo-3-chloropropane (DBCP) for the control of the citrus nematode (*Tylenchulus semipenetrans* Cobb) in established citrus groves. Control of the citrus nematode with emulsifiable DBCP in irrigation water was first reported by Reynolds and O'Bannon (2). They found that from 2 to 10 gallons/acre in 5 acre-inches of water effectively controlled the nematode around living trees in Arizona. More recently Baines, et al. (1) obtained satisfactory nematode control to 3-6 feet when the material was applied in basin irrigation but not with low pressure sprinkler irrigation. The application of DBCP in the irrigation water around perennial plants is preferable to injection, providing nematode control is comparable. The advantages of water application are usually lower application costs, more area treated, and less disturbance of tree roots.

With this in mind, several methods of application of emulsifiable DBCP were studied using the onion test (3) as a rapid method for determining the distribution of DBCP in soil 7 days after application.

SPRINKLER APPLICATION

Three 10-acre blocks of citrus were treated with 2.5 to 3.0 gallons/acre (active material) emulsifiable DBCP[2] using overhead rainbirds, each covering a 50-foot radius. The sprinklers were staggered on a diamond pattern. The water was allowed to run 12 to 15 hours, which delivered between 3 and 4 acre-inches of water. The soil from the trunk of one tree to the trunk of the opposite tree was covered with a polyethylene tarp 25 feet wide to provide an untreated area. The plots were sampled in three different positions (row middle, drip line, under tree) in order to determine the distribution of the fumigant in relation to the tree. Four tree sites per treated plot were sampled. The results are given in Table 1.

The distribution of DBCP was uniform in the row middle and drip line, however no DBCP was detected in any of the samples taken under the tree. The control of the citrus nematode paralleled the reduction in growth of the onion seedlings, however, better control due to diffusion of the fumigant should have been obtained, especially at the lower depths. Poor diffusion may have been due to the large amount of organic fertilizer and debris observed in the first foot of soil. Objections to the overhead sprinkler application were that the area under the tree was not treated, distribution of the fumigant was irregular, and control of the citrus nematode was erratic.

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[2]Trade name for material used was Nemagon, 25% by volume, produced by Shell Chemical Corporation.

Table 1. Percentage reduction in growth of onion seedlings 7 days after treatment, and control of citrus larvae 4 months after treatment with emulsifiable DBCP at the rate of 2.5-2.7 gallons/acre in 3-4 acre-inches of water through overhead sprinklers.

Sample location	% reduction of onion seedlings 0-1[a]	1-2	Citrus larvae per 50 cc soil: Treated 0-1[a]	1-2	2-3	Check 0-1	1-2	2-3
			PLOT I					
Row middle	30	20	0	0	0	30	170	30
Drip line	30	23	0	15	35	435	270	130
Under tree	0	0	790	970	--	1350	740	--
			PLOT II					
Row middle	34	32	0	3	680	5	10	180
Drip line	13	13	7	1200	450	1200	170	--
Under tree	0	0	270	300	1170	490	850	1120
			PLOT III					
Row middle	45	0	0	5	3	16	200	62
Drip line	43	0	0	30	240	1300	380	200
Under tree	0	0	500	170	230	270	187	--

[a]Sample depth in feet.

FURROW IRRIGATION

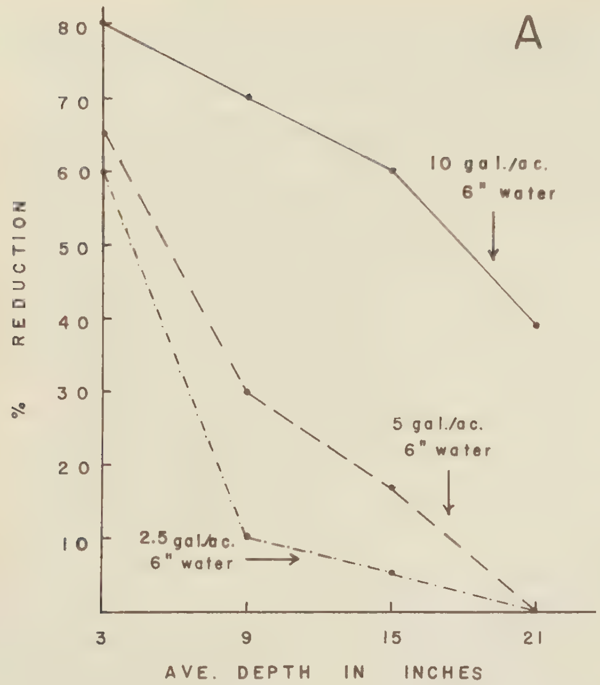
Two fields of different soil texture were selected to compare two formulations of emulsifiable DBCP[3] during furrow irrigation. According to the manufacturers, the only difference between these two fumigants was in their emulsifier. The "soluble 50" emulsifier was formulated to keep the DBCP in solution longer than regular emulsifiable DBCP. Plot I was a loam soil of 16% clay, 35% silt, and 49% sand, while Plot II contained 31% clay, 54% silt, and 15% sand. The fumigant was introduced by gravity into the water at the headstand, which passed through a 6-inch pipe with a baffle plate, and then distributed through six furrows on both sides of one row of trees. Approximately 4 gallons/acre DBCP (active material) was applied in 6 to 8 acre-inches of water over 22 hours in Plot I, and approximately 5 gallons/acre (active material) was applied in 17 acre-inches over 48 hours in Plot II. Tree rows were 20 trees long (360 feet) and were sampled at the 2nd, 9th, and 17th trees. Results of these tests are given in Table 2.

Table 2. Percentage reduction in growth of onion seedlings 7 days after treatment, and control of citrus nematode 4 months after treatment with two emulsifiable formulations of DBCP on two soils applied by furrow irrigation.

Treatment (active DBCP)	Location	Furrow no. 1 Av. % reduction in onion seedlings: 0-6[a]	6-12	12-18	18-24	Av. no. citrus larvae/50 cc soil, Furrow no. 1	2	3	untreated
				PLOT I		(0-3 feet)			
4 gallons/acre	beginning	56	58	60	61	0	0	0	940
Soluble 50 in	middle	56	58	58	55	8	56	901	1660
7.2 acre-inches	end	50	61	55	45	8	1151	1210	500
4 gallons/acre	beginning	61	55	53	40	0	0	0	
Regular Emul-	middle	50	60	58	61	0	58	785	
sifiable in 7.2	end	56	56	58	61	0	0	362	
acre-inches				PLOT II		Furrow no. 1 (0-2 feet)			
5 gallons/acre	beginning	62	48	0	0	2			440
Soluble 50 in	middle	60	10	0	0	150			1060
17 acre-inches	end	62	10	0	0	250			800
5 gallons/acre	beginning	66	10	0	0	175			
Regular Emul-	middle	60	4	0	0	275			
sifiable in 17 acre-inches	end	56	0	0	0	142			

[a]Sample depth in inches.

[3]Trade names for these fumigants are Soluble 50 Nemagon supplied by Oasis Chemical Co., and Emulsifiable Nemagon, supplied by Shell Chemical Company.



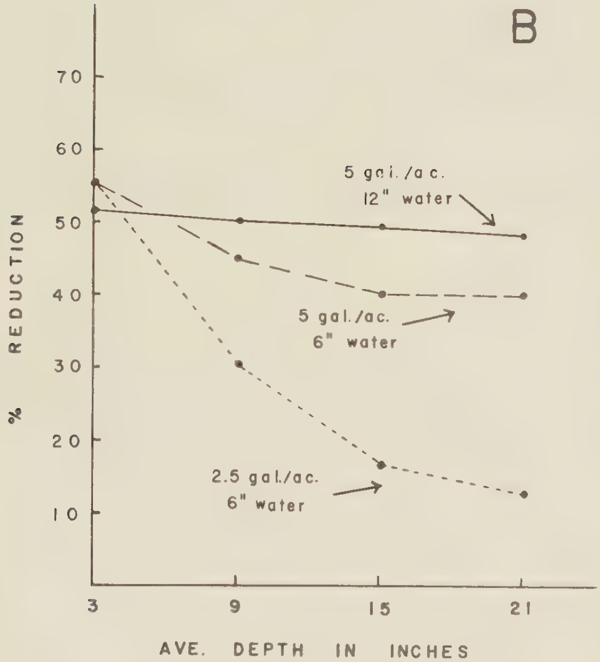


FIGURE 1. A -- Distribution pattern of 2.5, 5, and 10 gallons/acre of emulsifiable DBCP applied in 6 acre-inches of water measured by the reduction in growth of onion seedlings.

B -- A comparison of the distribution patterns of 2.5 and 5 gallons/acre of emulsifiable DBCP applied in 6 acre-inches of water and of 5 gallons/acre in 12 inches of water.

Plot I was an open, coarse-textured soil and took water rather rapidly. Results from the onion test indicate that there was a uniform distribution of the DBCP in furrow 1. Observations at application indicated that furrow 1 required appreciably more water flow than the total flow in furrows 2 and 3 (row middle). Thus, furrow 1 received more than half of the total DBCP applied, instead of the intended 1/3 of the total. This was substantiated by the nematode control data in which the nematodes were controlled to 3 feet in furrow 1, but only at the beginning of the furrow in furrows 2 and 3. This was probably due in part to the uptake of more water at the head than the end of the furrow.

The fine-textured soil of Plot II took water very slowly and soon sealed off, so that most of the water was running off before completion of the irrigation. The results of the onion tests indicate that the DBCP was only distributed in the top 6 inches of soil. There was only a slight reduction in the nematodes in the first two feet of soil (Table 2). Even though the dosage of fumigant was increased, the distribution and control was poor, probably due to the filtering out of DBCP on the clay and silt particles. No significant differences in control were observed between the two emulsifiable formulations used.

In the coarse-textured soil, DBCP was distributed uniformly in furrows provided the infiltration of water was uniform throughout the treated area. This, however, is the exception rather than the rule, in established citrus groves. Application of emulsifiable DBCP in furrows on the fine-textured soils did not give satisfactory nematode control.

BASIN IRRIGATION

Tests were conducted in metal basins 44 inches in diameter (3), and in earth basins, to determine what effect different rates and different amounts of water had on the distribution of DBCP[4].

The distribution of 2.5, 5, and 10 gallons/acre (active material) of emulsifiable DBCP in 6 acre-inches of water were compared on a fine-textured soil of 27% clay, 41% silt, and 32% sand. The organic matter content was 2.7%. The application of the fumigant was replicated three times in metal basins. The results of the onion test are presented in Figure 1A. The distribution of 10 gallons/acre was considerably better than 5 gallons/acre, however,

[4]Trade name for material used was Nemagon, 50% by volume, produced by Shell Chemical Corporation.

Table 3. Control of citrus nematode with emulsifiable DBCP applied in metal basins.

Treatment (gallons/acre)	Water (in inches)	Average number citrus larvae/50 cc soil (sample depth in feet) 0-1	1-2	2-3	3-4
		PLOT I			
Check		1525	1473	435	318
2.5	6	6	2196	148	786
5	6	0	468	346	842
10	6	0	0	30	130
		PLOT II			
Check		2242	2807	2335	1302
2.5	6	0	0	41	321
5	6	0	0	0	2
5	12	0	0	0	0

Table 4. Reduction in growth of onion seedlings 7 days after treatment, and control of the citrus nematode 4 months after treatment with 10 gallons/acre of DBCP (active material).

Sample depth (in feet)	Average % reduction in onion seedlings: 18 inches water	30 inches water	Average number citrus larvae/50 cc soil: 18 inches water	30 inches water	Check
0-1	59	56			
1-2	58	55 (0-3 ft.)	25	32	2420
2-3	61	46			
3-4	35	34	299	332	500
4-5	25	14	307	170	67
5-6	0	5	327	114	138

insufficient water was applied to obtain uniform distribution. Control of the citrus nematode was poor, due to shallow distribution of fumigant at application time, and sorbtion of fumigant on clay and silt particles, and organic matter.

In another field of 20% clay, 24% silt, and 56% sand, and rather low organic matter, 2.5 and 5 gallons/acre (active material) of emulsifiable DBCP was applied in 6 acre-inches of water. A third treatment consisted of applying the material in 6 acre-inches of water and then as soon as the solutions had infiltrated, applying another 6 acre-inches of water. Increasing the amount of water apparently resulted in a uniform distribution of DBCP throughout the top 2 feet of soil (Fig. 1B). The increased amount of DBCP in the 18- to 24-inch zone was also correlated with better nematode control (Table 3), at the 3- to 4-foot depth.

A test plot was set up to see how far 10 gallons/acre (active material) DBCP would penetrate in 18 and 30 acre-inches of water. Each treatment consisted of four soil basins around orange trees and each treatment replicated twice. Sampling was done by taking one soil core from each basin and compositing four samples per treatment. The DBCP was applied in the following manner: 7 gallons in 6 acre-inches of water + 6 acre-inches of water + 3 gallons in 6 acre-inches of water, and 7 gallons in 12 acre-inches of water + 6 acre-inches of water + 3 gallons in 12 acre-inches of water. The checks had corresponding amounts of water.

Results of these tests are given in Table 4. The onion tests indicated that the DBCP was uniformly distributed to a depth of 3 feet and then tapered off gradually to 5 feet. Although it appeared that sufficient material was distributed in the soil, nematode control was not so good as expected. This soil was high in clay and silt (17% clay, 40% silt, and 43% sand) and between 2 and 3% organic matter, which may have sorbed the chemical and reduced control of the nematodes.

Basin application of emulsifiable DBCP gave the most uniform distribution of the fumigant in the soil. Other advantages of this method of application include treatment of the entire area around the trees and the use of large amounts of water when needed.

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PRESERVATION AND GERMINABILITY OF CONIDIA OF PERONOSPORA EFFUSA (GREV. EX DESM.) CES.

Muriel J. O'Brien and Raymon E. Webb[1]

Summary

Germinability studies of preserved blue mold conidia were conducted over a 12-month period. Infection was obtained with conidia from stored leaves during an 8-month period.

Defrosting experiments demonstrate that great care should be taken to avoid defrosting and refreezing inoculum during the storage period and just prior to removal of inoculum from the frozen state for screening experiments.

Decrease in germinability of blue mold conidia begins promptly after storage of infected spinach leaves at -10° F and continues at a progressive rate with increased length of storage time.

INTRODUCTION

To screen new introductions and segregating populations of spinach, *Spinacia oleracea*, for resistance to blue mold, *Peronospora effusa* (Grev. ex Desm.) Ces., blue mold-infected leaves held at -10° F are used as inoculum sources. The method of inoculum preservation (1) has been described.

Additional aspects of preservation and germinability of blue mold conidia were investigated during the past year: 1) the effect of defrosting and re-storage of the infected leaves on spore germination; 2) the germinability of conidia held for as long as 1 year at -10° F; and 3) infectivity of conidia during an 8-month period.

MATERIALS AND METHODS

Spinach leaves abundantly covered with blue mold conidia were collected from greenhouse-inoculated plants, placed in screw-top glass food jars, and stored immediately after collection at -10° F.

To investigate the effect of defrosting and re-storage of infected leaves on spore germination, the leaves were stored for 72 hours at -10° F and then defrosted at room temperature for 1, 2, and 5 minutes and completely defrosted. The defrosted leaves were then re-stored at -10° for 24 hours, after which time the germination counts were made.

In the long-term preservation study blue mold-infected leaves were stored at -10° F for 1 year and germination counts were made at bimonthly intervals. These infected, stored leaves were also used as inoculum source in the infectivity study.

In making germination counts the blue mold spores were brushed from the hard-frozen leaves with a 00-size sable-hair artist's brush into 50-ml beakers containing a small amount of distilled water. The suspensions were then held at 56° F for 24 to 72 hours. Commencing at 16 to 24 hours germination counts were made. Germination percentages are based on numbers of total germinated spores in 10 microscopic fields (43X) in each of six drops of suspension on the slide.

RESULTS AND DISCUSSION

The conidia from all stored samples germinated directly by lateral germ tubes. Rate of germination was slower and length of germ tubes was shorter in conidia from stored leaves.

Preliminary testing indicated that defrosting and refreezing of infected spinach leaves reduced germinability of blue mold spores. The data in the defrosting experiment show that germinability loss increases with each lengthening of the defrosting period (Table 1); the greatest loss occurred in the spores harvested from completely defrosted leaves. Therefore, in utilizing preserved inoculum great care should be taken to control defrosting conditions before collection of spores from the frozen leaves. Sharp freezing, with the unbroken maintenance of a hard-frozen state, ensures greater germinability of the spores.

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Table 1. Germination of blue mold conidia from sharp-frozen infected spinach leaves held at -10° F and subjected to defrosting and refreezing.

Period of defrosting before refreezing (minutes)	Number of spores counted	% spores germinated[a]
0	1231	15.6
1	1228	14.8
2	1193	14.1
5	1109	11.2
9[b]	1060	9.8

[a]Just before storage -- 16.8% germination.
[b]Complete defrosting.

Table 2. Germination of blue mold conidia from sharp-frozen infected spinach leaves held at -10° F for 1 year.

Period in storage (months)	Number of spores counted	% spores germinated
0[a]	1186	18.5
2	1476	5.2
4	1403	3.8
6	1364	2.6
8	1179	1.8
10	1334	0.4
12	1205	0.8

[a]Prestorage count.

Table 3. Germination of blue mold conidia from sharp-frozen infected spinach leaves held at -10° F during a 30-day storage period.

Period in storage	Number of spores counted	% spores germinated
0[a] hours	1177	8.8
1	1300	7.4
2	1296	7.3
4	1124	9.4
8	1219	4.7
24	1054	4.2
48	1081	5.7
96	1051	6.3
8 days	1099	4.5
15	1115	5.0
30	1303	3.5

[a]Prestorage count.

The trend noticed in the defrosting experiments turned our attention to the effect of unbroken, hard-frozen storage on germinability of spores stored for extended periods of time. Germination percentages (Table 2), based on bimonthly counts of blue mold spores, show greatest loss in germination occurred within the first 2 months of storage and a continuance of germination decline to the end of the storage period. At the end of the 12-month storage period percentage germination was extremely low.

The sharp decline in germination between initial and 2 months' storage prompted an investigation of spore germinability at an hourly to daily basis up to 30 days. In this 30-day study a constant but not drastic loss of germinability occurred from the initial storage time through the entire month (Table 3). After 15 days there was a sharper decline in germination.

Infection was obtained with conidia from leaves stored for 2, 4, 6, and 8 months. No major difference was noted in infection potential among the samples stored for the varying lengths of time. No tests of infectivity of stored inoculum were made after 8 months because further testing was not feasible due to difficulty in maintaining proper greenhouse temperatures. However, on the basis of infectivity of spores stored 8 months, it is expected that infection could be obtained with conidia stored for extended periods provided tremendous spore loads are used. Ordinarily, periods required for extended storage of inoculum would not be in excess of 6 months, or 2 seasons in the temperate zone, because of the ease with which the fungus could be carried on living plants during the cooler seasons.

This method of sharp freezing spinach leaves covered with freshly-produced conidia of blue mold offers an opportunity to explore the possible existence of additional races of the organism (2). Often infected leaves of spinach hybrids and varieties known not to be susceptible to blue mold are received. Preservation of inoculum until proper screening conditions and diagnostic varietal reactions could be worked out would be of great help in screening spinach varieties suspected of being infected with new races. Also, preservation of inoculum over unfavorable testing periods ensures continuity of inoculum source for use in a spinach disease-resistance breeding program.

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DECAY OF ARTICHOKE BRACTS INOCULATED WITH SPORES OF BOTRYTIS CINEREA FR. AT VARIOUS CONSTANT TEMPERATURES

Werner J. Lipton and John M. Harvey[1]

Abstract

The rate at which artichoke bracts decay after inoculation with spores of *Botrytis cinerea* increases with temperature, but not uniformly, in the range of 0° to 20° C. The effect of temperature on the rate of decay is greatest during the early period of incubation. Infections studied almost invariably started at the tip of the bract and rarely at the point of abscission from the fleshy base. Bracts with injured tips decayed more rapidly than uninjured bracts, the difference in rate in some tests being equivalent to a difference of 10° C in holding temperature. Avoidance of injury and maintenance of low temperature would reduce appreciably decay of globe artichokes during marketing.

INTRODUCTION

Botrytis cinerea Fr. has long been recognized as the major cause of decay of artichokes (*Cynara scolymus*) during marketing. Link, et al. (1) described the disease in detail and pointed out that certain strains of the fungus grew in culture at temperatures as low as -2° C, had optimum growth at 22° to 25° and little growth above 31°. Pathogenicity studies indicated that about the same critical temperature relationship applied to growth of the fungus on inoculated buds.

Injury to the bracts favored infection, according to studies by Link, et al. (1) and by Rappaport and Watada (2). No quantitative comparisons were made, however, to assess the influence of injury on decay development. The effect of temperature on the rate of decay was not determined. Knowledge of the relation between temperature, injury, and decay is essential to the development of methods to control the disease during transit and marketing of the buds.

The relation of temperature and injury to decay was studied in the harvest seasons of 1957-58 and 1958-59. Artichokes were obtained near Castroville, California, and were transported to the laboratory at Fresno, California. Because about 8 hours were required to collect the buds and transport them to Fresno, they were held overnight (at 4° C) before starting the tests.

METHODS

Test 1 (December 1957): Temperature effects were studied on buds that were free from mechanical injury or visible decay. Sound-appearing bracts from the second and third complete whorl of the buds were broken off and surface sterilized by dipping for 30 seconds in a solution of 5000 ppm sodium ortho-phenylphenate (Dowicide A) and rinsed for 5 minutes in running tap water. The bracts were inoculated by dipping in a suspension of *B. cinerea* spores and were incubated in sterile dishes at high humidity. Four replicates of four bracts each were held at temperatures of 0°, 5°, 15°, and 20° C for various lengths of time.

Test 2 (March 1958): A further study of temperature effects was made with apparently uninjured bracts from the fourth to sixth complete whorl of the buds. Since these bracts were assumed to be relatively free of contamination, they were not surface sterilized, but were inoculated directly by dipping in a suspension of *B. cinerea* spores in a water solution of 0.05% Tween-20 (polyoxyethylene sorbitan monolaurate). Preliminary tests had shown that this spreader did not inhibit the infection of artichoke bracts by *Botrytis*. The bracts, replicated as in test 1, were incubated at 0°, 5°, 10°, and 15° C.

Test 3 (December 1958) and Test 4 (January 1959): Injury effects were studied in two tests, one started on December 11, 1958, the other on January 6, 1959. Artichokes from the same lot were used in both tests and were held at 0° C in the interval. In each test uninjured bracts from the fourth to sixth whorl of a bud were separated into two lots. The bracts in one lot were injured at the tip by pinching with a forceps. Both uninjured and injured bracts were inoculated with Botrytis spores in a water suspension, and one bract from each treatment was placed in

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an incubation jar. Three replicates (jars) were incubated at 5°, 10°, and 15°.

Examinations: The number of examinations and the interval between them varied with the incubation temperature. The extent of the decay was estimated visually and expressed as a percentage of the bract area that was decayed at each examination.

RESULTS AND DISCUSSION

Rate of Decay: The time required for decay to affect 1% of the area of the bract was considered to be the time required for an infection to become established. The time required for 10 or 20% decay to develop was a measure of the rate of enlargement of the affected area.

Both the time required to establish an infection (Fig. 1) and the time required for a given percentage decay (Fig. 2) decreased as the temperatures were increased. At 0° C more than 1 month was required for 1% of the area of a bract to decay. This interval greatly exceeded the normal marketing period for artichokes. At 10°, however, only 10 days were required for 1% decay to develop. Since the interval between harvest in California and consumption in eastern markets is about 2 weeks, the importance of holding artichokes below 10° C during all phases of handling is apparent.

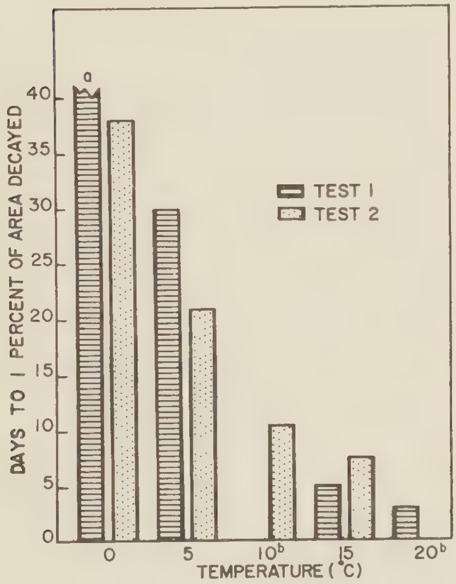
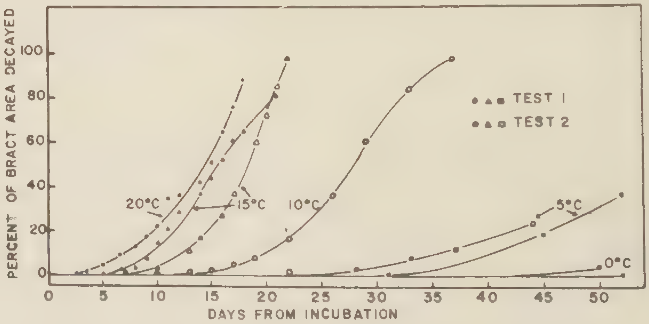


FIGURE 1. The effect of temperature on the time (days) required for 1% of the area of individual artichoke bracts to decay after inoculation with spores of B. cinerea.
[a]After 52 days only about 0.6% of the area of the bract was decayed.
[b]Only indicated test run at this temperature.

FIGURE 2. The development of decay in individual artichoke bracts inoculated with spores of B. cinerea during holding at various constant temperatures.



Decay, once established, does not spread at a constant rate at a given temperature, but rather at a continually increasing rate until about one-half or more of the bract is decayed (Fig. 2). The point where the rate begins to decrease is largely governed by the shape of the bracts, since the advancing margin of the decayed area narrows as it approaches the base of the bract.

The effect of temperature on rate of decay is greatest during the early stages of incubation. In test 1, about 40, 9.5, and 7 days elapsed between the time of inoculation and the development of decay in 10% of the area of the bracts held at 5°, 15°, and 20° C, respectively; the time intervals between the 10 and 20% levels of decay were 5, 2, and 2 days at these temperatures. In test 2, the corresponding time periods were 35, 20, and 13 days for the initial period and 7, 3, and 2 days for the latter period at 5°, 10°, and 15°, respectively. Low temperature is effective in retarding infection, but it is much less effective in slowing decay if the organism has become well established. Prevention of infection appears, therefore, to be the most promising method of reducing decay in artichokes.

Table 1. Decay of injured and uninjured artichoke bracts inoculated with spores of B. cinerea and held at 5°, 10°, or 15°C.

Test 3 December 1958

Observation	Treatment at °C					
	5°		10°		15°	
	I[a]	NI[b]	I	NI	I	NI
Days to visible decay	6	-[c]	3	8	1	7
Days to 10% decay	12	-[c]	7	17.5	3	10.5

Test 4 January 1959

Observation	Treatment at °C					
	5°		10°		15°	
	I[a]	NI[b]	I	NI	I	NI
Days to visible decay	9	10.5	3	5.5	0.5	2
Days to 10% decay	11	13.0	5.5	11	3.5	5.5

[a]I = tips injured by pinching.
[b]NI = tips not injured.
[c]In excess of 18 days, end of experiment.

Studies on the effect of injury on the development of decay (tests 3 and 4) showed that injury is a major factor contributing to rapid decay (Table 1) as was suggested earlier by Link, et al. (1). In the first run (December), uninjured, inoculated bracts held at 15°C decayed less rapidly than injured ones at 10° and uninjured bracts at 10° decayed less rapidly than injured ones at 5°.

In the second run in January similar relationships were found, but the rate of decay was generally more rapid. The buds were probably more susceptible to decay after being held for almost a month before testing.

OBSERVATIONS ON SITE OF INFECTION

Infections almost invariably started at the tip of the bracts and decay progressed basipetally, even when the tips were not visibly injured. Link, et al. (1) made similar observations on entire buds and noted that the exposed tips of the buds were probably injured during handling, or possibly even before harvest. Microscopic examination of apparently uninjured spines frequently revealed small cracks in the surface that might serve as ports of entry to the pathogen. In contrast, the large wound made at the point of abscission of the bract from the receptacle was rarely the site of infection. The exudate from the broken vascular tissue and the wounded cells of the fleshy base would seem to provide an ideal opening for infection, but the reason for its relative immunity is unknown.

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OCCURRENCE OF HADROMYCOSIS IN LOUISIANA COTTON

John P. Hollis

Previous occurrences in Louisiana cotton of hadromycosis caused by Verticillium albo-atrum Reinke & Berth. have been light and sporadic. Records exist for five parishes: Caddo, Bossier, and Red River in the upper Red River Valley of northwest Louisiana and East Carroll and Tensas in the Mississippi Delta section of northeast Louisiana[1].

During the last week in August 1960 the inception of a severe outbreak involving 220 acres of cotton was noted on the Clyde Smith farm in Rapides Parish in the Red River Valley of central Louisiana. The acreage was comprised of three varieties: DPL 15-130, Stardel-60, Auburn 56-30, on soils ranging from Yahola sandy loam to Miller clay, but classified primarily as Yahola silt loam. The disease developed rapidly during a 3-week period and on September 14 approximately 50% of plants of the DPL 15 variety exhibited symptoms. High proportions of Stardel and Auburn 56 plants were also affected. Distribution of diseased plants was more or less random but small aggregates of affected plants frequently occurred in a single row.

The disease was characterized first by a slightly lighter color in terminal leaves. This was followed by the occurrence of single large yellow blotches on 2-3 leaves at random, terminating in general wilting, dying of the blotched areas and severe defoliation of the plant. The vascular symptoms consisted of elongated, light-brown streaks oriented longitudinally and occurring throughout the xylem region at the base of the stem and extending throughout the shoot system as a light brown discoloration of the wood. Only a trace of root-knot nematode infestation was found in any of the cotton varieties.

Isolation of the fungus was effected by incubation of pieces of streaked wood in sterile water in Petri plates. Examinations after 5 to 7 days revealed a characteristic gray mycelium with the verticillate sporulation habit of the causal fungus. There were no microsclerotia.

Verticillium hadromycosis is commonly distinguished in Louisiana from Fusarium wilt by:

1. late season occurrence
2. prevalence on relatively heavy soils
3. scattered pattern of diseased plant distribution
4. susceptibility of all cotton varieties, including Plains and Auburn 56, grown for resistance to Fusarium wilt.

This is the first report of severe hadromycosis in cotton in Louisiana and the first instance of its occurrence in Rapides Parish.

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PRELIMINARY REPORT ON NEMIC PARASITES OF COFFEE IN GUATEMALA, WITH SUGGESTED AD INTERIM CONTROL MEASURES

B. G. Chitwood[1] and C. A. Berger[2]

Abstract

A brief review of reports of nemic parasites and associates of coffee is given; 14 genera and 20 species are listed. Of these, 12 genera and 17 species are considered of pathogenic significance for some type of agricultural crops; 5 genera and 9 species are thought to be potential pathogens of coffee. Meloidogyne exigua s.l., M. incognita s.l., and Pratylenchus coffeae are reported as severe pathogens of coffee in Guatemala. Coffea arabica Bourbon and C. robusta are both affected, though the former is more injured than the latter. Ditylenchus procerus and Helicotylenchus erythrinae are reported as being suspected of pathogenic significance to coffee in Guatemala. Symptoms of the first three species are illustrated. Sugarcane and native field corn are reported as detrimental if immediately preceding coffee planting. A program of horticultural control methods is proposed for these pathogens.

INTRODUCTION

This is a very preliminary report on observations made primarily by the senior author but including a small amount of comments on or/and information acquired by others both prior to and since his association with the problems of the Asociación Experimental Cafetalera. These other observations and facts may or may not all have been verified by both or either of the present authors. They are included in order to put information on record, to stimulate others to assist or assume responsibility for phases of the further investigations apparently necessary, and to provide the coffee plantations of Guatemala with at least the minimum advisory assistance they may need while a more satisfactory long-term control program of the nemic and associated problems affecting coffee production in Guatemala is developed.

The authors feel that the finer details of the pathologic side of the investigation can well be temporarily omitted and that those phases of the problems encountered which clearly appear to be either purely theoretical, or without economic significance, can well be postponed until such time as they are apt to have a direct immediate bearing on the development of control measures that should increase crop production.

HISTORICAL RÉSUMÉ

A brief historical résumé of nemic diseases of coffee on a world-wide basis and their control will, for the most part, have to be delayed for the present. An extremely brief summary is presented by Cramer (8). Those facts on record which appear to have an immediate bearing on the development of a sound program will be included in the present article, so far as time and space limitations permit.

Members of the Nemata (=Nematoda sensu restricto et sensu lato) previously and presently reported as of possible significance to coffee production include:

1a. Meloidogyne exigua exigua E. A. Goeldi, 1887 (11). Brasilian pyroid coffee nema. First reported by that author as a cause of major damage to Coffea (variety not stated), "Province" of Rio de Janeiro, Brasil. Presumably meant by Schieber and Sosa (31) in reporting on symptoms and resistance of "C. arabica arabica, C. arabica bourbon, C. arabica pache and C. robusta" from some Guatemalan plantations. (Possibly = 1d)

b. Meloidogyne sp. L. R. Krusberg & H. Hirschmann, 1958. Pyroid coffee nema. Reported from samples from "The Selva," around Tingo Maria, Peru as a possible cause of damage in 4 of 12 samples from "Coffee." Bally and Reydon (2) reported on "Heterodera radicicola" as a cause of major damage to Javanese coffee seedlings which exhibited primary root cortex breaking.

c. Meloidogyne africana africana A. G. Whitehead, 1959 (36). Whitehead's pyroid coffee nema. First reported by that author as a cause of major damage to Coffea arabica L. (variety

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not stated), Meru district of Kenya, East Africa.

d. Meloidogyne exigua sensu lato B. G. Chitwood & C. Berger, 1960. Brasiloid pyroid coffee nema. Reported as a cause of major damage to Coffea arabica L. v. Bourbon and C. robusta L. (less damaged) on several estates in Guatemala. (See 1a)

e. Meloidogyne incognita sensu lato B. G. Chitwood & C. Berger, 1960. Southernoid pyroid coffee nema. Reported as contributory to damage of C. arabica L. v. Bourbon, on some estates in Guatemala.

f. Meloidogyne inornata L. G. E. Lordello, 1956 (24). Mentioned as tentative but unconfirmed identification by G. Thorne in E. Schieber and O. N. Sosa (31). (Possibly = 1e)

2a. Pratylenchus coffea (A. Zimmermann, 1898) (37) T. Goodey, 1951 (17). Javanese lesion coffee nema. Reported as a cause of damage to C. arabica L. in Java and since in other countries. C. robusta and C. uganda also infected. Much work has been done. Reported by Schieber and Sosa (31) from Guatemala. Also reported as cause of root damage and stunting of C. arabica var. Bourbon and C. robusta in Guatemala by Chitwood and Berger (4).

b. Pratylenchus brachyurus (G. H. Godfrey, 1929) (10) T. Goodey, 1951. Godfrey's lesion nema. Reported by L. R. Krusberg and H. Hirschmann (23) from "Coffee" roots, Peru.

3a. Helicotylenchus erythrinae (A. Zimmermann, 1904) M. Golden, 1956 (13). Javanese spiral nema. First reported as possibly causing damage to Coffea spp. Java, since reported as endemic in most coffee-producing areas of the world. Reported by Chitwood and Berger (4) from Guatemala.

4a. Radopholus similis (N. A. Cobb, 1893) (6) G. Thorne, 1949 (34). Fijian burrowing banana nema. First reported major cause of damage to coffee by Zimmermann (38). Since found as a major cause of decline in coffee plantations over much of the earth's surface. Not found in Guatemala.

5a. Ditylenchus procerus (W. Bally & G. A. Reydon, 1931) I. N. Filipjev, 1936 (9). Javanese (coffee) root eelworm or nema. Reported as of questionable pathogenic significance by original authors from roots of Javanese coffee. Reported by Chitwood and Berger (4) from C. arabica v. Bourbon in Guatemala.

6a. Paratylenchus macrophallos (J. G. deMan, 1880) (27) T. Goodey, 1934 (15). Large penis pin nema. First reported by Krusberg and Hirschmann (23), from roots of "Coffee" from Peru. Recently reported as rare in, and probably not of pathogenic significance to, coffee in Guatemala by Chitwood and Berger (4).

7a. Xiphinema americanum N. A. Cobb, 1913 (7). American dagger nema. Known as a severe pathogen of some crops. Reported from about Peruvian coffee roots by Krusberg and Hirschmann (23), and as a rarity, probably of no pathogenic significance to Guatemalan coffee by Chitwood and Berger (4).

b. X. radicicola T. Goodey, 1936 (16). Goodey's dagger nema. Known as an ectoparasitic root pathogen of some plants. Reported from about Peruvian "Coffee" roots by Krusberg and Hirschmann (23).

8a. Trichodorus christiei M. W. Allen, 1957 (1). Christie's stubby root nema. Known as a severe pathogen of some plants. Reported as about the roots of Peruvian "Coffee" by Krusberg and Hirschmann (23). Also reported as a rarity, probably of no pathogenic significance to Guatemalan coffee by Chitwood and Berger (4).

b. T. monohystera J. W. Seinhorst, 1954 (32). Seinhorst's stubby root nema. Reported as about Peruvian "Coffee" roots by Krusberg and Hirschmann (23).

9a. Tylenchorhynchus sp. Stunt nema. Known as a pathogen in roots of some plants. Reported from roots of Peruvian "Coffee" by Krusberg and Hirschmann (23).

10a. Rotylenchus sp. Spiral nemas. Known as pathogen in roots of some plants. Reported by Krusberg and Hirschmann (23) from roots of Peruvian "Coffee."

11a. Criconemoides sp. Ring nemas. Known as pathogen in roots of some plants. Reported from roots of Peruvian "Coffee" by Krusberg and Hirschmann (23).

12a. Hemicriconemoides sp. Sheathoid nemas. Suspected pathogens of roots of some plants. Reported by Krusberg and Hirschmann (23) from Peruvian "Coffee" roots.

13a. Aphelenchoides coffeae (A. Zimmermann, 1898) G. Steiner, 1937 (33). Javanese coffee root eelworm or nema. Reported as a suspected pathogen of coffee by Zimmermann (39). Since often recorded as a suspect. Reported as endemic and prevalent but probably a fungivorous form in coffee roots, and not significant to coffee production, unless as a vector of other diseases or supposed non-pathogenic fungi, by Chitwood and Berger (4).

b. Aphelenchoides bicaudatus (S. Imamura, 1931) (20) T. Goodey, 1951. Bicaudate nema or eelworm. Suspected as a possible pathogen of some crops but reported as a prevalent endemic, but fungivorous, form in Guatemalan coffee roots by Chitwood and Berger (4). (See 13a)

14a. Aphelenchus avenae H. C. Bastian, 1865 (3). Bastian's aphelench. Often reported as a suspected pathogen of various plant roots and reported as a fungivorous species, not of pathogenic significance unless as a vector. Reported as endemic in and about Guatemalan coffee roots by Chitwood and Berger (4).

OBSERVATIONS

1. Pyroid nemas (Meloidogyne) similar to, but slightly different from, the Brasilian pyroid coffee nema and the Southern pyroid Lovell nema, were observed attacking coffee roots on some plantations in Guatemala. The former species was a major limiting factor to C. arabica Bourbon production on several estates. Symptoms included lower stem and root galling with surface breakage, severe stunting, chlorosis, and even death of the trees. Roots exhibited cracking and galling. Seedlings in nurseries are extremely stunted, the chlorotic leaves often drop and the plants die. Older trees, even mature ones, may be killed. External lower stem galling and root cracking provides an entry for all types of other organisms including normally saprophytic fungi. The extent to which this is developed may depend partly on massive infections of young seedlings at transplanting, and partly, on the hot and humid climate. Sugarcane is a bad immediate predecessor for coffee planting wherever Brasiloid pyroid coffee nemas are present. Southernoid pyroid coffee nemas cause more damage when native field corn precedes coffee planting. However, they do not appear to cause the extensive root cortex cracking that the Brasiloid coffee nema does, nor does the stem galling appear to become quite so advanced. Southernoid pyroid coffee nemas also cause damage to C. robusta seedlings and minor damage to mature trees of this variety. For comparative purposes a photograph of a few feeding roots of C. arabica Mundo Nova attacked by the Brasilian coffee pyroid nema is presented (Fig. G). The extent to which the differences in symptoms are due to differences in the nemic taxa and to differences in the host varieties cannot be guessed in this case.

2. Javanese lesion coffee nemas (Pratylenchus coffeae) cause all of the same above ground symptoms as do pyroid nemas (Meloidogyne) except stem galling. Cases in which they were definitely the predominant pathogenic organism, when seedlings were killed, were numerous. Due to the prevalence of mixed infections, it was impossible to be sure whether they also caused root swellings and root cortex sloughing. Field laboratory examinations caused us to suspect there might be a second type of lesion nema involved, but this was never verified. Extensive instances of root cortex sloughing (Fig. F) caused us to suspect the presence of burrowing nemas (Radopholus similis) but not a single specimen was encountered.

3. The Javanese spiral nema (Helicotylenchus erythrinae) was extremely abundant on the roots of C. arabica Bourbon on all plantations visited, but due to the coincident presence of either pyroid nemas or lesion nemas in every instance, the damage attributable to the spiral nemas could not be assessed.

5. Perhaps the most interesting and puzzling nemic pathogen encountered was the Javanese coffee nema or eelworm. The incidence and populations of these organisms were as high as those of pyroid, lesion and spiral nemas and they commonly occurred in root swellings. However, due to the prevalence of pyroid and/or lesion nemas in the same plant roots, one could not be certain that the former species actually caused root swellings or growth reduction. The fact that Greeff's root gall eelworm or nema (Ditylenchus radicicola (R. Greeff, 1872) (18) I. N. Filipjev, 1936), which is congeneric, is known to cause root galling in grasses (14), with consequent growth reduction, causes the Javanese coffee root eelworm (D. procerus) also to be placed on the highly suspect pathogen list, at this time.

6-8. Rare occurrence of pin, dagger and stubby root nemas in small numbers in, or about, the roots of C. arabica at the plantations visited, caused the observers to doubt that the particular species encountered had any significance to Guatemalan coffee production, though they are known to be major pathogens of some plants in other parts of the world.

13-14. Javanese coffee root nema, or eelworm (Aphelenchoides coffeae) and the bicaudate nema (Aphelenchoides bicaudatus) have both been suspected by others to cause damage to cultivated plants by invading the roots. The former species was even considered as a highly suspect pathogen of coffee in Java, while the latter species was considered as a suspected pathogen of rice in Japan. Our observations indicate these are both fungivorous species, as is Bastian's aphelench (Aphelenchus avenae), and though all three species commonly invade coffee roots, are endemic, and may be present in considerable numbers in coffee root tissues, they may well be ignored, unless they are shown to injure the coffee by providing an entry means, and acting as a vector for other damaging organisms. We do not feel they should be taken seriously, since their food appears to be limited to fungi. Even supposedly non-pathogenic fungi may become established in the coffee roots by these same organisms.

FIGURE 1. See opposite page for legend.

SUGGESTED AD INTERIM COFFEE PRODUCTION CONTROL PROGRAM

From the standpoint of increasing coffee crop production during the period in which better, more economical, and more lasting methods are developed, the authors of this article make the following tentative suggestions:

1. Coffee seed and coffee seedlings should not be planted on land or in soil immediately following sugarcane or native field corn, unless measures have been taken to destroy, or greatly reduce, any known and any seriously suspect nemic pathogens of coffee.
2. Seedlings exhibiting gross (visual) symptoms of such pathogens should be discarded or treated in a manner that specialists consider as sufficiently effective.
3. Only apparently healthy and vigorous seedlings should be transplanted and used, in any way, for propagatory purposes.
4. An effort should be made to determine the extent of qualitative and quantitative crop reduction, the part the individual kinds of pathogens may play, and the extent of the possible roles of injurious complexes, in the limitation of crop production. Fertilizers and horticultural practices, local climates and conditions, host ranges, prevalence, and virulence of pathogenic organisms should be considered.
5. From the standpoint of control of nemic pathogens the following tentative suggestions are put forward:

(a) The presently growing practice of trying Crotalaria spp. as shade and green manure in seed beds, nurseries, and intended coffee production plantings might well be encouraged and extended (26). This might include trials of other species of Crotalaria such as C. spectabilis Roth. which is supposed to act as an effective ground cover, trap and control crop for members of some of the genera of known nemic pathogens of coffee.

Dr. C. L. Horn of Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland has suggested that C. vogelii Benth., the stems and leaves of which also are known to be a source of rotenone, could conceivably provide a second income crop in addition to acting as a source of nitrogen (green manure). It would be advisable to test the efficiency of these species in increasing coffee crop production through the control of nemic pathogens (25).

(b) Recent observations that Asparagus officinalis L. may be nonspecifically effective in reducing nemic plant pathogens and also produce an additional income crop seem to warrant exploration (29).

(c) Marigolds (Tagetes spp.) were reported as "resistant" to "root knot" as early as 1915 (28) and as early as 1936 (12). Comparative field tests of marigold species were conducted in efforts to determine differences in their "suitability" as hosts of nemic pathogens. Recently

FIGURE 1. A. Coffea arabica var. Bourbon, 6 year, "El Carmen," Guatemala. Brasiloid pyroid coffee nema. 1.4X. Sugarcane previously; no roots free of symptoms. Stem and tap root galling is extreme with secondary fungi and other organisms predominant. Causal pyroid nemas difficult to locate.

B. Another example of stem galling as a result of Brasiloid pyroid coffee nema. 1.4X. The cortex of both stem and roots is cheesy. Main tap roots and secondaries are involved, often destroyed.

C. C. arabica Bourbon from "Tambor," Guatemala. 1.4X. Stem galling and root galling as caused by the southernoid pyroid coffee nema is not quite as pronounced as in A-B and root surface cracking apparently does not occur as a result of this pathogen.

D. C. arabica Bourbon from "El Carmen," Guatemala. Roots 1.4X. Note extreme root proliferation, some stellate galling as a result of stimulating production of new feeder roots. Swollen roots near tip and browning of roots in galled areas is common.

E. C. robusta, 30-year-old tree with roots infected with southernoid pyroid coffee nema. 0.61X. "El Carmen," Guatemala. Note swollen root tip.

F. C. arabica Bourbon, 2-year-old, infected with Javanese lesion coffee nema following sugarcane. 0.61X. "El Carmen," Guatemala. Note some root browning and swelling with tendency toward cortex sloughing.

G. C. arabica Mundo Novo, attacked by the Brasilian pyroid coffee nema. 1.4X. Origin São Paulo, Brasil. Root swellings are elongate or spheroid, bloated appearing, commonly brown. Minor differences from A-F may be due to biotype of pathogen or to host varietal differences. Specimen kindly supplied by Dr. Luiz Gonzaga E. Lordello, Escola Superior de Agricultura "Luiz de Queiroz," Universidade de São Paulo, Brasil.

[Magnifications for all figures reduced by approximately 25%.]

(1956 - 1959) Tagetes patula L. and T. erecta L. have been found to be effective in reducing soil populations of additional nemic pathogens and such results may be due to production by their roots of non-specific nematicides (35). Field observations on native marigold species, microscopic examinations of their roots, artificial inoculation tests with known nemic pathogens of coffee, similar laboratory and field studies relative to the possible use of rank growing non-endemic species, with a view toward their possible use as ground covers for infected coffee plantings, would seem warranted.

(d) Tripsacum guatemalensis (otherwise known as T. laxum Nash and T. dactyloides (L) L.) (=Gamagrass) has been successfully used as a green manure, and ground cover (19), and we have been informed that recently it has been used successfully in alleviation of damage caused by pyroid nemic pathogens of various tropical crops, but we have not as yet located a published record to this effect.

(e) Festuca elatior L. var. Kentucky 21 (=Tall or Meadow Fescue), according to personal communication (J. N. Sasser, Department of Plant Pathology, North Carolina State College, Raleigh, North Carolina), has been found to be an effective ground cover and non-host of at least 5 pyroid nemic biotypes.

(f) Other possible plants which might be found to be economically sound as ground covers, even though their "suitability" as hosts to biotypes of known nemic pathogens of coffee and/or other plants may appear to be specific, might well be explored from this standpoint. Such plants include Arachis hypogaea L., Fragaria spp. crosses, Capsicum frutescens L. vars., Lycopersicum peruvianum (L.) Mill., Avena sativa L. and other "Key hosts" of pyroid nemas (5, 21, and 30) or lesion nemas.

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HOST-PARASITE INTERACTIONS OF THE ASIATIC PYROID CITRUS NEMA

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Abstract

The host range and gross pathology of Asiatic pyroid citrus nemas in Taiwan is reported and illustrated. Such organisms are also reported from New Delhi. Unless they constitute a mixture which is suspected, they differ from all other known pyroid nemas (better known as "root-knot nematodes"). The natural infections were on Citrus reticulata austera and C. sinensis v. ?, respectively, while successful transfers were made from the former host to C. sinensis "Sha Kan" and "Wango," C. grandis, "Wanbanyu," and C. aurantium "Chu Lan," Zea mays Kao Kwang, Ipomoea batatas v. loc., and Sorghum vulgare v. Piper Sudan Grass. "Unsuitable" hosts in which galls were formed and damage may or may not have occurred included Fragaria ananassa "Red Rich" and "Furi," Arachis hypogaea v. loc., Avena sativa Minhafer, Glycine max Whip-Poor-Will, Capsicum frutescens v. loc. and "California Wonder," Camellia sinensis v. ?, and Coffea arabica v. loc. In the latter the larvae did not develop beyond the second stage. Lycopersicum esculentum "Marglobe" and "Rutgers" permitted development to adulthood and jelly masses were produced but no eggs or larvae have yet been observed. In all instances in which the specimens reached adulthood the females broke the root surface and jelly masses were produced external to the root. Horticultural control measures are suggested.

INTRODUCTION

The earliest report of pyroid nemas parasitizing citrus (commonly called "root-knot nematodes") was that of Neal (1889 Bull. U.S. Div. Ent. No. 20, 31 pp., 21 pls.) who reported (p. 11) the "Bitter sweet orange," Citrus vulgaris, and the orange and lemon etc., Citrus aurantium var. sp. as slightly affected by Anguillula arenaria J. C. Neal, 1889 for which the type host Arachis hypogaea L. was later designated by Chitwood (1949, p. 98, Proc. Helminthol. Soc. Wash. D. C. 16: 90-104, figs. 1A-6F) when it was transferred to the genus Meloidogyne. Subsequent investigators have not been able to verify this finding as regards any biotype of the genus presently known in the U.S.A.

This article is to place on record certain information relative to the host range and gross pathology of the pyroid nema found parasitizing citrus in New Delhi and Taiwan (Chitwood and Toung, 1960, Phytopathology 50: 631-632) for which we propose the vernacular name Asiatic Pyroid Citrus Nema. Assuming this does not consist of a mixture of biotypes it is the most morphologically variable taxon of the genus Meloidogyne yet reported. However, the present host transfer records must be repeated on the basis of replicate single egg mass isolation stocks and the various stages studied morphologically before it would be proper to give it, or them, a scientific (Latin) name. In the meantime interesting peculiarities of the host-parasite relationships can be put on record and we make a few minor suggestions relative to ad interim control measures.

A full analysis of previous reports of "root-knot nematodes" from citrus in other parts of the world, following Neal's original report, will have to be delayed for the present. However, the references which we have thus far located are as follows:

N. A. Cobb (1890, Agr. Gaz. New South Wales 1(2): 155-184, figs. II-6VI) quotes W. Easson May 19, 1890 letter, (p. 185) as stating that roots of orange trees are not troubled by "root-knot" in New South Wales.

G. Lavergne (1901a Publ. Estac. Patol. Vejetal Chile No. 9 (Febraro) (Estracto del Diario; del Boletin de la Sociedad Nacional de Agricultura), 12 pp., figs. 1-2) named a root parasitic organism Anguillula vialae (p. 6) from "viñedos de Talca, Quillota, Mellipilla, etc. stating the same organism existed in Limoneros; Naranjos de Coyanco (p. 9). Second and third publications by the same author (1901b Rev. Chilena Hist. Nat. 5(4) (Abril): 85-91, figs. 8-9, pl. 3) and (1901c Rev. de Viticulture 8 Année 16(410) (October 26, Paris): 445-452, figs. 75-83) appeared. In the last article, at least, there appears to have been some confusion of organisms which will

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require extended study. There is no question, however, but that Lavergne was dealing with at least one member of the genus Meloidogyne in these articles.

H. J. Webber and W. A. Orton (1902, U.S. Bur. Plant Industry Bull. No. 17: 23-36, pls. V-VI) reported careful examination of orange roots from all parts of Florida disclosed no trace of "root-knot" injury.

E. A. Bessey (1911, U.S. Dept. Agr., Bur. Plant Industry Bull. No. 217: 1-89, table I, figs. 1-3, pl. I, fig. 1-pl. III, fig. 2) confirms H. J. Webber and personal communications from P. H. Rolfs in this regard (p. 11).

G. Gandera (1920, Sec. de Agr. y Fomento, Dir. Agr. Bol. (n.s.) 111, 40 pp., illus. (Mexico)) reported "Heterodera radicicola" "un anélido microscópico" on Citrus aurantium at a ranch in Yucatan.

M. Jean Ghesquière (1921, Bull. Agr. Congo Belge 12(4) (Decembre): 709-718, figs. 239-251) reported Tylenchus (Heterodera) radicicola on the roots of several species of Citrus (p. 710) from Eala, Belgian Congo.

According to Tyler (1941, U.S. Dept. Agr. Misc. Publ. No. 406, 91 pp., pp. 8 and 23 cited) the Nematode Committee, California Agr. Exp. Sta. (unpublished data, 1925?) reported Citrus roots resistant (to root-knot).

J. R. Watson and C. C. Goff (1937, Florida Agr. Exp. Sta. Bull. No. 311, 22 pp., tables 1-3) reported Citrus trees seem to be entirely free from attack (by root-knot nematodes).

L. R. Krusberg and H. Hirschmann (1958, Plant Disease Reptr. 42: 599-608, fig. 1, table I, p. 603 cited) reported finding only larvae of Meloidogyne sp. in 3 of 11 samples of Citrus roots from "The Selva" in Pêru.

In order to discuss the (or these) organisms parasitizing citrus roots in Taiwan and New Delhi, we are proposing the vernacular name Asiatic Pyroid Citrus Nema, pending its (or their) allocation to definite species of Meloidogyne.

In the following report on the host range and gross pathology of the pyroid nemic pathogens of citrus the genera of hosts are indicated by arabic numerals and the species and varieties of hosts by italicized latin letters. A plus (+) sign precedes the arabic numeral or/and the italicized letter designating the variety in those cases in which the life cycle of the nema or nemas was successfully completed; a minus (-) sign, similarly, precedes the numeral or letter in those cases in which the life cycle was not completed; and a plus or minus (±) sign will be used in those cases in which the males and females reached maturity but no eggs were observed. The notation Nat. infect. signifies natural infection; Art. infect., signifies artificial infection; field, indicates the test host was planted under an infected citrus tree; pot, indicates the test host was planted in steam sterilized soil and infected citrus roots added to the soil; pot EM, indicates that the test host was planted in steam sterilized soil and egg masses from infected citrus roots were added; transpl., indicates the test host was transplanted from other areas; transpl. treated, indicates the test host was treated for Meloidogyne before being transplanted; and control indicates control test host plants were also used; seed, indicates the test hosts were sprouted from seed planted in steam sterilized soil; cut., indicates the test hosts were rooted cuttings; TW, indicates Taiwan observations; and Calif. indicates California observations; d = days. In some cases the expression "10 (20 or 30) g citrus root inoc." appears. This means that heavily infected citrus feeding roots were collected, chopped, washed, drained dry and the stated quantity used as inoculum. Since the two authors have worked independently, at long distance, the independent observations of each are preserved in this manner and any observations by one author not as yet confirmed by the other must remain in some minor doubt. We hope to be in a position to repeat this work in a more refined manner at a later date and to conduct the more refined microscopic pathologic investigations found desirable as a result of these preliminary crude observations. Due to the urgency of the problem we feel that placing the present information on record will be of some value. The suggested ad interim control measures are the responsibility of the senior author, as is the presentation of this article.

OBSERVATIONS

+1a Citrus reticulata Blanco var. austera Swing. (Fig. A). (Mandarin Orange). Nat. infect., preliminary descr. with some illustrations of nema submitted Nov. 12, 1959 to the senior author by junior author as a new species designating this as type host, Yangmingshan, Taipei, China, as type locality, the date of discovery being recorded as Nov. 2, 1959.

+1b Citrus sinensis (L.) Osbeck var. (Fig. B). (Sweet Orange). Nat. infect. roots were simultaneously submitted by Dr. S. M. Chatterji, Entomology Research Institue, New Delhi, India.

Calif. observations indicated the organisms were of the same type. In both cases the feed-

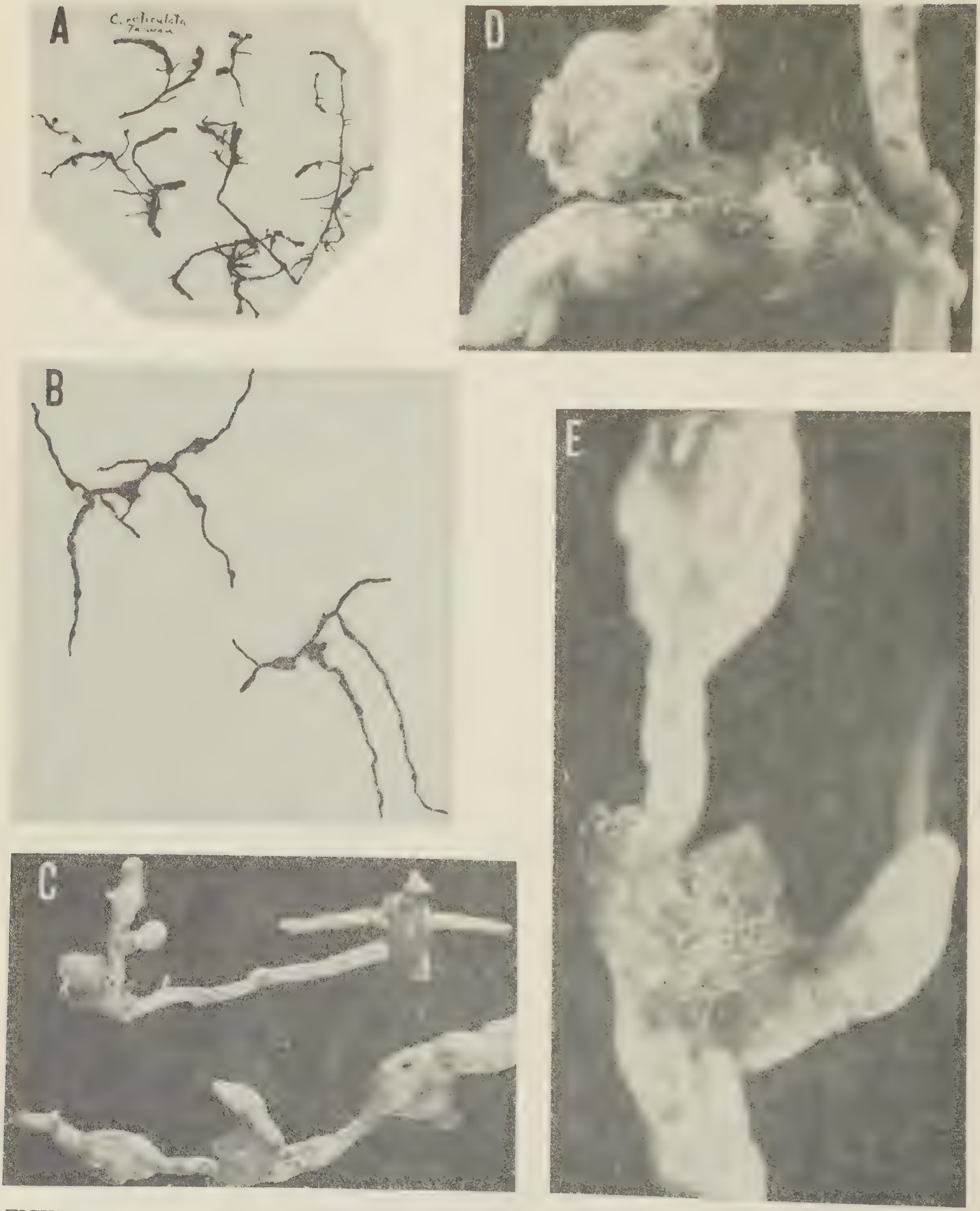

FIGURE 1. See opposite page for legend.

ing roots exhibited elongate to discrete enlargements, root tips somewhat bloated, minor areas of necrotic discoloration (browning) along rootlets, including tip browning.

+1c C. sinensis "Sha Kan". (Figs. C-E). Art. infect., field, seed, transpl. Comments-- explanation of figs.

+1d C. sinensis "Wango". Art. infect., field, seed, transpl.

+1e C. grandis (L.) Osbeck "Wanbanyu" (Pummelo). Art. infect., field, seed, transpl.

+1f C. aurantium L. "Chu Lan" (Sour Orange). Art. infect., field, seed, transpl.

+2a Zea mays L. "Kao Kwang" (Figs. F-G). (Corn). Art. infect., field, seed, transpl., TW 30d, leaves yellow, growth stunted; Calif., some rounded root galls, late larval males and females, no adults, eggs or second stage larvae; 49d, Art. infect., field, seed, transpl.; TW leaves deep yellow, growth stunted, many distinct galls, egg masses, with many larvae.

+3a Ipomoea batatas Lam. var. local. (Fig. H). (Sweetpotato). Art. infect., pot, cut. transpl., 10 g citrus root inoc. TW 48d: some pale yellow leaves, growth stunted. Calif.: many discrete root galls, life cycle completed, egg masses external; secondary roots enlarged from 1 to 2 mm diam.; occasional root tip blinding.

+4a Sorghum vulgare Pers. Piper Sudan Grass. (Sorghum Grass). (1) Art. infect., pot, seed, transpl., 10 g citrus root inoc. TW 30d, 2nd 10 g citrus root inoc. (top growing well); 58d, some yellow leaves, growth stunted. Control, root and top normal. Calif.: many minute root galls, males, females and external egg masses. Life cycle completed. (2) Art. infect., field, seed, transpl. TW 59d: many distinct galls and some egg masses, more than 10 larvae 1 egg mass; some light leaves, growth stunted.

-5a Fragaria ananassa Hort. "Red Rich" (Strawberry). Art. infect., field, transpl. (not treated). Calif.: 30d: Meloidogyne hapla. Test invalid.

-5b F. ananassa Hort. "Furi" (not clear whether this represents another name for same variety). (1) Art. infect., pot, transpl., treated, 30 g citrus root inoc. TW 31d: minute galls, top grew normally; control, no galls, top normal; 63d: same as 31d. (2) Art. infect., field, transpl., treated. TW 31d and 62d: root and top normal.

-6a Arachis hypogaea L. var. local. (Domestic Peanut). (1) Art. infect., pot, seed, transpl., 10 g citrus root inoc. TW 57d: some yellow leaves, no distinct galls or egg masses, some swellings and brown roots. Control pot: root and top normal. (2) Art. infect., field, seed, transpl. TW 57d: many leaves became yellow, some dropped; growth stunted; no distinct galls or egg masses.

-7a Avena sativa L. Minhafer. (Oat). Art. infect. (1) pot, seed, 10 g citrus root inoc. TW 57d: growth nearly normal; some pale yellow leaves; 3 visible galls and some brown roots; no egg masses. Control pot: root and top normal. (2) field, seed, transpl. TW 57d: growth stunted, many pale yellow leaves; 5 visible galls and many brown roots; 79d: as 57d, but many leaves had dropped.

-8a Glycine max (L.) Merr. Whip-Poor-Will. (Soybean). Art. infect. (1) pot, seed, 10 g citrus root inoc. TW 30d: many leaves yellow, dropped; new leaves while older had dropped, continuous process; visible galls. Control pot: top and root as in inoc. pot. TW 40d: inoc. and control seedlings dead. (2) field, transpl. 3 seedlings. TW 30d: top and roots as in (1); TW 40d. All dead. Attempts with this variety discontinued in Taiwan.

-9a Capsicum frutescens L. local variety. Fig. K. (Red Pepper). Art. infect., field, seed, transpl. 2 large seedlings. TW 20d: many chlorotic leaves, new leaves shrunken; after several d. normal leaves chlorotic, new shrunken leaves sprouted, such continued 57d. Calif. 57d

FIGURE 1. Asiatic pyroid citrus nema. (The color statements relative to these photographs were prepared by A. Blaker, Photographer, Life Sciences Bldg., University of California, Berkeley, who took the pictures.)

A. Citrus reticulata austera. Natural infection. Yangmingshan, Taipei, Taiwan. Feeder roots natural size. Several external egg masses, excessive root branching, galls both discrete and elongate.

B. C. sinensis var. ? New Delhi, India. 1.37X. Note egg masses also external and galls much the same in form. Minor differences in symptoms from A are probably due to host variety response or quantity of larvae attacking roots.

C. C. sinensis var. "Sha Kan" seedling artificial inoculation grown c. 90 days. 7.4X. Note brown root lesions, parasitized appearance of host tips, and external egg masses. One female apparently fallen out of root gall to left; second female exposed.

D. C. sinensis var. "Sha Kan." 24X. Numerous small brown lesions, two jelly masses and female exposed by root breaking.

E. C. sinensis var. "Sha Kan." 24X. Note bloated appearing root tip and eggs within jelly mass. [Magnifications for all figures reduced by approximately 25%.]

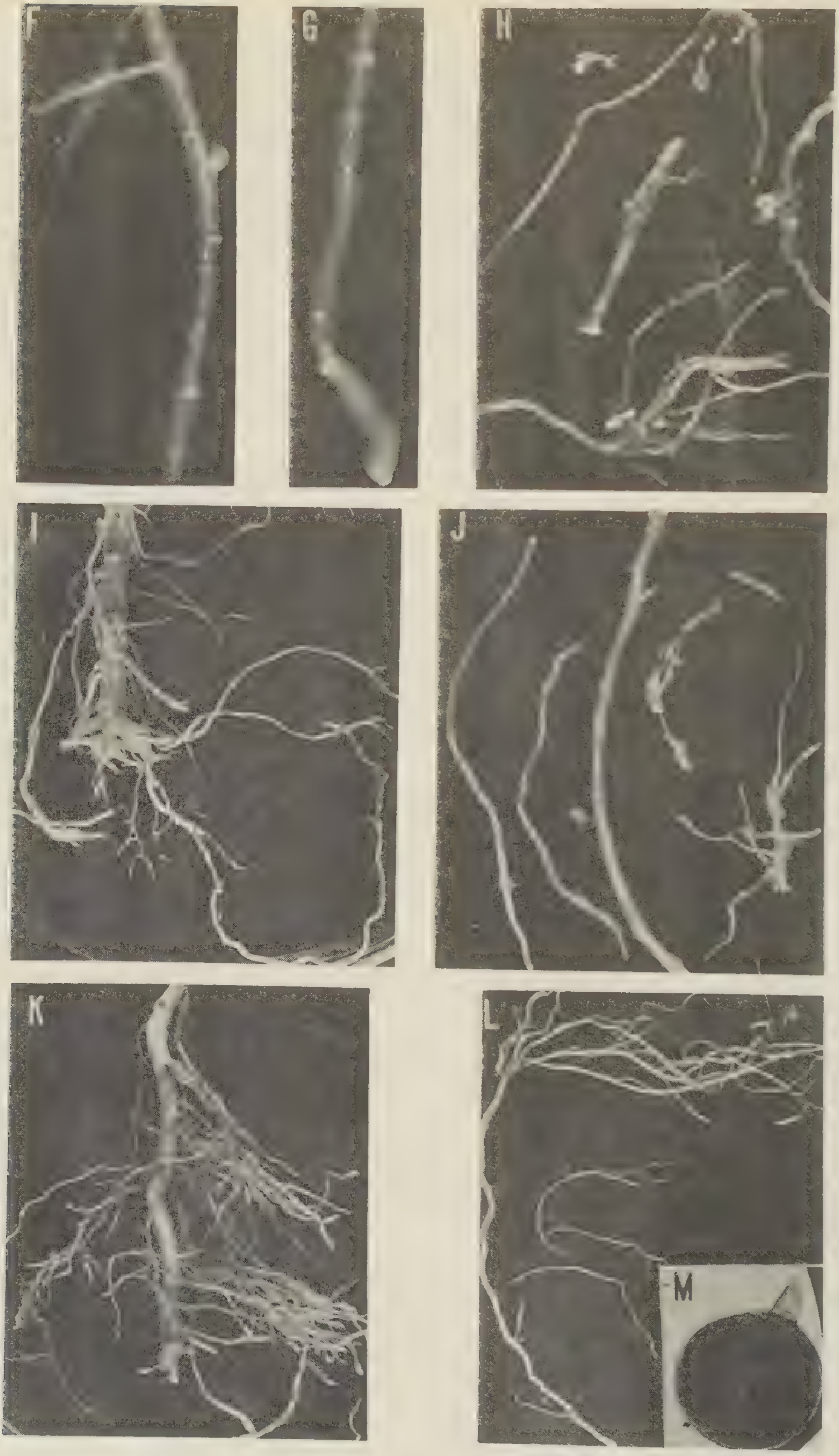

FIGURE 1. (continued) See opposite page for legend.

root galls very rare and indistinct, only 1 discrete gall; root cap and tip browning; no nemic rests on dissection. (2) transpl. seedling to pot (sterilized soil) TW to 77d: top recovery day by day; 97d: top completely normal; many new roots, no new galls, no new brown roots. (3) pot, seed, 10 g citrus root inoc. TW 20d: some chlorotic leaves; new leaves shrunken. TW 40d: top became normal day by day. 57d: top completely normal; no distinct galls, some root swellings and brown roots. Control pot: 57d: top and root normal.

- 9b C. frutescens L. California Wonder. Art. infect., pot, seed, 10 g citrus root inoc. TW 35d: some chlorotic leaves, new shrunken leaves; no visible root galls. TW 64d: top normal; some root swellings, no distinct galls or egg masses. Control pot: top and roots grew normally.

- 10a Coffea arabica L. var. Unknown. (Fig. L). (Arabian Coffee) Art. infect. (1) field, seed, transpl. TW 29d: top growing well. Calif. 34d: few elongate small root galls on dissection only dead 2nd stage larvae. (2) pot, 10 g citrus root inoc. TW 69d: top grew very well; some root galls, no egg masses. Control pot: top grew very well; no root galls. (3) field, seedling, transpl. TW 69d: top grew very well; 12 small galls, no egg masses.

- 11a Camellia sinensis (L.) O. Kuntze var. Unknown. (Domestic Tea). (1) Art. inoc., pot, seed, 10 g citrus root inoc. TW 20d: no galls ("It might be due to no new roots sprouting and larvae could not enter roots"). TW 30d: many new roots; 10 g citrus root (2nd inoc.). TW 65d: top normal; minute galls seen with hand lens. Control pot: top and root normal. (2) Art. inoc., field, seed, transpl. TW from (1) after 30d. TW 30d. No galls on roots. field, transpl. TW 105d (total): top normal; many minute galls, no egg masses.

± 12a Lycopersicum esculentum (L.) Mill. Marglobe. (Domestic Tomato). Art. inoc. (1) field, transpl. from field. TW 30d: root galls present. Calif.: M. incognita. (2) pot, seed, transpl. 3 single EM inoc.'s: 2 with no root galls, 1 with 3 galls and 1 minute jelly mass. TW 47d: latter, no additional galls; TW 62d: original 3 galls disappeared, no new galls; top normal, many brown root pits. Control pot: no galls or brown root pits. (3) pot, 10 g citrus root inoc.: TW 20d: some light, some deep yellow leaves; growth stopped. To TW 50d: seedling recovered day by day, new shoots sprouted; TW 62d: older roots mostly brown and many swellings but no distinct galls or jelly masses; new roots white. Control pot: top and root normal. (4) field, seedling, transpl. TW 29d: many deep yellow leaves, some leaves dropped; growth severely stunted; no distinct galls. Calif. 30d (Figs. I-J): adult males, females, minute jelly masses,

FIGURE 1 (continued).

F. Zea mays var. Kao Kwang. 9X. Shows numerous females just under root surface and breaking through, and an external egg mass.

G. Zea mays var. Kao Kwang. 9X. Root tip enlarged and with slight yellowing female breaking through root surface slightly proximal to tip.

H. Ipomoea batatas var. local, Taiwan. 4X. The small root fragment upper right was externally yellowed; one external egg mass and one female is exposed. Upper piece shows an exposed female. Two abnormal lateral branch roots both with tip enlarged, one greatly, and darkly browned; the entire root exhibited medium browning; center root fragment, yellowing with medium and dark browning near root tip, two egg masses are exposed; far right fragment, yellowed with brown near center exhibits individual eggs distinguishable in the egg mass, and nearby a female which has broken through the root surface is exposed. Lower fragment exhibited yellowing throughout, with tip browning of rootlets and dark browning of the center multi-female section where the excessive root proliferation is obvious. Root cortex breakage and sloughing appears to follow heavy infection in this host. This is an obviously satisfactory host.

I. Lycopersicum esculentum var. Marglobe. 2X. 32 days after inoculation. Roots were slightly yellowed throughout with brown lesions and even cups on stem indicating necrosis. This may be a case in which the nemic enzymes are so destructive to the plant tissues on which they feed that the cells die and the nemas are deprived of nourishment. This plant was transplanted from steam-sterilized soil.

J. Same as I. 4X. Far left piece, browned upper end. Upper center piece browned. Second from left piece, extensively browned in moderate tones. Second from right, browned in areas which appear dark in photo. Far right piece, browned in areas dark in photos.

K. Capsicum frutescens var. local. 2X. Yellowing with deep tip browning; moderate browning on lower stem.

L. C. arabica var. local. 2X. Yellowed throughout with some individual rootlets rather darkly browned over the entire length; brown lesions on main stem.

M. Female, Asiatic pyroid citrus nema. 68X.

[Magnifications for all figures reduced by approximately 45%]

no eggs or 2nd stage larvae; roots somewhat yellowed throughout length, brown lesions and even cups, presumably indicating position from which adult females were dislodged when roots were washed. (5) field, seedling, (sterilized soil) transpl. 63d old. TW 62d after transpl.: many deep yellow leaves, some leaves dropped; growth severely stunted; roots mostly brown, 9 distinct galls, 3 minute jelly masses; no larvae. To 82d seedling declined, some new shoots sprouted, did not grow normally. TW 90d: many new roots, no new distinct galls, no jelly masses.

+12b L. esculentum (L.) Mill. Rutgers. Art. inoc. (1) 10 pots each single EM inoc. TW 54d: tops normal; each pot some roots swellings and brown pittings. (2) pot, 20 g citrus root inoc. TW 20d: leaves light, pale yellow; growth stopped. TW 61d: many pale yellow leaves, some leaves dropped; many brown roots, 4 distinct galls, no egg masses. Control pot: top and root normal. (3) field, transpl. from source of (2). TW 55d: many pale yellow leaves, some leaves dropped; many brown roots, 6 distinct galls but no egg masses.

DISCUSSION

From these observations it would appear that the Asiatic pyroid citrus nema is physiologically more virulent than most of the other pyroid nemic biotypes described, since it causes such extensive necrosis even on "hosts" in which it does not reproduce. The actual micropathology will be investigated at a later date. Another outstanding characteristic is that in the case of all "satisfactory hosts" the egg masses are produced externally. In the hosts studied, the females do not usually assume as pyroid a gross outline as is common for other biotypes of pyroid nemas in most of the hosts in which they reproduce (Fig. M).

Whether or not this biotype can be induced to reproduce on Marglobe or Rutgers tomatoes by proper adjustment of fertilizer, temperature etc. is a point yet to be determined. Since we are approaching the completion of the life cycle so closely on these hosts, it is difficult for us to believe that it cannot be completed, with proper adjustments. If not, it will certainly be an example of unusually peculiar type of partial "resistance."

SUGGESTED AD INTERIM CONTROL PROGRAM

Finally, while awaiting the development of satisfactory long-term economic control measures for this organism in citrus, we would like to suggest that the growers in Taiwan consider the use of:

(1) Crotalaria as a cover crop in the citrus orchards or groves. See McKee, R., G. E. Ritchie, J. L. Stephens, and H. W. Johnson. 1946. Crotalaria, Culture and Utilization. U.S. Dept. Agr. Farmers' Bull. No. 1980, 17 pp., 14 figs. Supt. Documents, Washington, D.C. Another reference which includes use of Crotalaria for pyroid nemic control is Watson, J. R., and C. C. Goff. 1937. Control of root-knot in Florida. University of Florida, Agr. Exp. Sta., Gainesville, Florida Bull. No. 311, 22 pp., tables 1-3.

Dr. Claude Horn of Plant Industry Station, U.S. Department of Agriculture, kindly called to our attention that one of the species of Crotalaria, namely C. vogelii Benth., has the additional value of being a good source of rotenone. Its possible use, and possible resistance to this particular biotype of pyroid nema, might well be considered from the standpoint of a possible supplemental income crop also acting as a good green manure and nemic poisoning crop.

In any proposed use of these hosts as a cover crop, the possible exposure of livestock to poisoning is an important consideration. The bionomic and agricultural place of the various Crotalaria species and their adaption to the climate of the area involved are important.

(2) The use of strawberries as ground cover and trap crop as well as a cash crop is certainly worthy of consideration.

(3) Peanuts, oats (var. Minhafer), soybeans (var. Whip-Poor-Will), and peppers as trap crops and ground covers would appear to be logical, if economically sound. These, as well as strawberries and Crotalaria might well be expected to stimulate citrus production in diseased groves. The legumes should also add nitrogenous materials, and all should aid as green manure.

(4) We understand that Guatemala grass Tripsacum guatemalensis (also known as T. dactyloides L.) has been used successfully in some parts of the world as a green manure in plantings where pyroid nemas are a problem. Of course, we have no information relative to its possible value when this biotype is involved but feel that it also should be considered.

Ref. Hitchcock, A. S. 1951. Manual of Grasses of the United States. 2nd. Rev. by A. Chase, U.S. Dept. Agr. Misc. Publ. No. 200 (p. 790) pp. 1051, figs. 1199. Supt. Documents, Washington, D.C.

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A BORON DEFICIENCY DIEBACK IN HIGHBUSH BLUEBERRY[1]

C. G. Woodbridge and Richard H. Drew

Abstract

In 1958, a dieback on highbush blueberries was noted on a farm in Western Washington. This condition was most prevalent on the Pioneer and Pemberton varieties. Stanley showed slight dwarfing. There was a high correlation between the low boron content of tissues and the incidence of the trouble. The condition is believed to have been caused by a low soil moisture content which allowed the affected plants to wilt in 1958. Boron was applied in 1958. There was no dieback in 1959 and 1960.

INTRODUCTION

In 1958 on a highbush blueberry farm in Western Washington, a dieback on some of the older and larger plants growing in localized areas was noticed. The soil is a sandy loam having a pH of 4.8 and is irrigated during the summer months. A review of the literature on blueberry culture did not suggest a possible cause. However, because of the suddenness of the appearance of the trouble and because the area in which the farm is located is known to be low in boron for some crops, it was deemed worthwhile to study this problem from its relationship to boron nutrition.

SYMPTOMS

Other workers have described an induced boron deficiency on blueberries growing in sand culture (3, 4). Both groups of workers noted a browning of the apical tissues of branches, followed by some dieback. Adjacent leaves on the affected branches became chlorotic, blotched, and misshapen. Sometimes red-brown areas appeared on the leaves. No mention was made of such a condition being found in fields of blueberry bushes nor have the authors come across any such report.

In the Washington farm, where the condition was prevalent, three varieties were affected. During the growing season of 1957 all bushes had made good growth and had appeared normal. However, in the spring of 1958, many buds, both leaf and fruit, failed to develop, especially on the Pioneer variety. Figure 1 shows a normal branch and one with dieback symptoms. On other bushes only a few branches were affected (Fig. 2), and on still others only a few buds failed to develop and sloughed off.

FIGURE 1. Pioneer variety: Branch on left shows typical dieback symptoms; branch on right is normal.

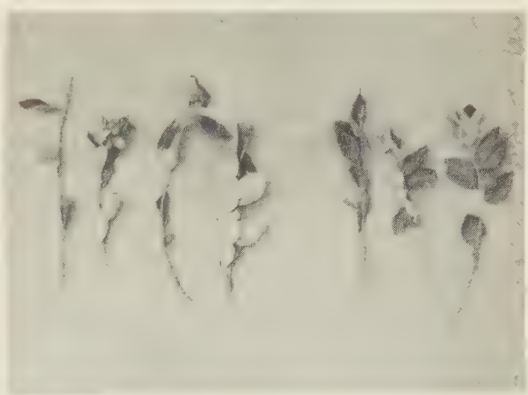

FIGURE 2. Pioneer variety: Branches on left show varying degrees of injury; some buds failed to develop and sloughed off. Normal branches on right.

[1]Scientific Paper No. 2021. Washington Agricultural Experiment Stations, Pullman, Washington. Project No. 9055.

The buds on the Pemberton variety broke normally, but after several weeks of apparently satisfactory growth the terminal bud shrivelled, turned brown, and died. This last symptom is somewhat similar to those described for plants growing in sand culture (3, 4). In some instances the terminal 2 to 3 inches of the shoot and the leaves also died.

The Stanley variety did not show any characteristic symptoms, but rather a general stunting and a marked lack of vigor of the entire plant.

Jersey showed no abnormalities.

EXPERIMENTAL

In September 1958, after the crop had been harvested, samples of leaves and twigs for chemical analysis were collected from four varieties (Pioneer, Pemberton, Stanley and Jersey) from bushes in and near the affected areas. The bushes in the affected area were 22 and 15 years old, whereas those in the non-affected area were about 15 and 10 years old. In sampling, no attempt was made to distinguish between fruiting leaves and twigs and sucker leaves and twigs. Samples from the Pioneer bushes had of necessity to be taken from primarily fruiting shoot wood as little new growth was available on the affected plants. The Pemberton samples were taken from the sucker wood since that was where the symptoms were most pronounced. Soil samples were obtained near the affected bushes and also from a non-affected area.

Samples were analysed for boron by the method of Dible et al. (2). Boron sprays (12 pounds of "Solubor" per acre) were applied to the affected bushes in the fall of 1958. No sprays or fertilizers containing boron had previously been applied.

RESULTS

Results of analyses for the boron content of leaves, twigs, and soils are presented in Table 1. There was no recurrence of the dieback in 1959 and 1960.

Table 1. The boron content of blueberry leaves, twigs, and soils[a].

Description	Variety			
	Pioneer	Pemberton	Stanley	Jersey
Leaves				
Affected area, severe symptoms	21.6	11.1	----	----
Affected area, normal	38.3	14.5	23.1	17.9
Non-affected area, normal	50.6	61.0	67.2	34.1
Twigs				
Affected area, severe symptoms	15.5	12.9	----	----
Affected area, normal	22.0	15.0	11.5	18.7
Non-affected area, normal	23.7	23.7	24.5	26.1
Soils				
Affected area	0.45	0.53	0.49	----
Non-affected area	0.65	0.43	0.35	0.39

[a]Results are expressed as ppm on a dry weight basis and for the plant tissues are the average of the results of analysis on separate samples from six plants. For the soils, results are the average of three determinations.

DISCUSSION

The results in Table 1 show that a very high correlation exists between the condition described and a low boron content of tissues from affected blueberry bushes. There is no correlation between soil boron and the condition. The results of analyses for soil boron suggest that there was most probably an adequate amount of this element present but that the bushes were unable to utilize it.

The sudden appearance of this trouble is difficult to explain unless it is assumed that the

dieback was caused by a lack of boron induced by low soil moisture. The blueberry farm is furrow-irrigated and only about one-seventh of the annual precipitation (45 inches) falls during the summer months. The 1957 spring and summer were exceptionally hot and dry and even with irrigation many of the bushes wilted from time to time. This wilting may have weakened or permanently damaged the 1958 buds while they were being formed. Boron deficiency in apple -- internal and external cork of fruit and dieback -- has been reported (1) as caused by summer drought. Blossom blast on pears (5) has also been shown to be associated with a low soil moisture content.

There appears to be a varietal difference in susceptibility to boron deficiency. The Pioneer variety showed the most marked symptoms, followed by Pemberton. Stanley showed only a lack of vigor, whereas Jersey showed no abnormalities even when growing in the same area where the other varieties were most severely affected.

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HEAT TREATMENTS TO DESTROY FUNGI IN INFECTED SEEDS AND SEEDLINGS OF CITRUS

L. J. Klotz, T. A. DeWolfe, C. N. Roistacher, E. M. Nauer, and J. B. Carpenter[1]

INTRODUCTION

Citrus fruits near or on the ground may become contaminated by Phytophthora spp., fungi that also destroy the bark of roots and stems of citrus trees (5). Seeds extracted from such fruits and used in the seedbed contaminate not only the seedbed, but subsequently the nursery and orchard planting sites. This report describes a hot-water seed treatment to eliminate this source of contamination.

REVIEW OF PREVIOUS INVESTIGATIONS

Baines, Klotz, Clarke, and DeWolfe (1) found that a 10-minute immersion in water at 116° F killed the citrus nematode, Tylenchulus semipenetrans Cobb, in roots and the common brown rot gummosis fungus, Phytophthora citrophthora (R. E. Sm. & E. H. Sm.) Leonian, in the trunk bark of bare-rooted sweet orange trees on sweet orange rootstocks. Exposures of 15 minutes or longer at that temperature injured the trees. With balled trees treated in moist air (60% relative humidity) at 110°, 16 to 20 hours were required to kill these parasites. Klotz, DeWolfe, and Wong (unpublished data) found that 9- to 15-month-old seedlings of the following varieties, when hardened by heeling in moist soil for 1 to 2 weeks before heat treatment, were not injured by a 10-minute immersion in water at 118°: Cleopatra mandarin, Citrus reticulata Blanco; Shangyuan (Ichang pummelo), C. ichangensis Swing x C. grandis (Linn.) Osbeck var.?; Rubidoux and Webber-Fawcett trifoliate oranges, Poncirus trifoliata (Linn.) Raf.; Sampson tangelo, C. paradisi Macfayden x C. reticulata var. Dancy; Troyer citrange, C. sinensis (Linn.) Osbeck x P. trifoliata; Jochimsen grapefruit, C. paradisi; Rough lemon, C. jambhiri Lushington; Jameson sweet orange, C. sinensis; and Standard sour orange, C. aurantium Linn. Later this treatment was found to disinfect seedling roots infected by P. citrophthora which produces no oospores. It failed to kill P. parasitica Dastur which does produce this spore form. Birchfield (2) in Florida showed that an exposure to 122° for 10 minutes was required to kill the burrowing nematode, Radopholus similis (Cobb) Thorne. Unfortunately, this treatment would be injurious to seedlings or nursery trees of some varieties. Also, as will be seen later in this paper, it would not be effective against the thick-walled oospores of P. parasitica, an important destroyer of the bark of roots and stems.

Klotz, DeWolfe, and Wong (6) found seeds of Troyer citrange, four varieties of trifoliate orange, sour orange, and sweet orange tolerant to a 4-minute immersion in water at 120° to 140° F without appreciable loss of germinability. Four minutes at 120° or above destroyed P. parasitica and P. citrophthora in infected seeds. These results indicated that the thick-walled heat-resistant oospores and other heat-tolerant spores, such as chlamydospores and sporangia of P. parasitica, were not present in these seeds. The hot-water immersion treatment of citrus seed was suggested, therefore, as a means for decreasing the risk of contaminating seed beds, nurseries, and field plantings on virgin or disinfested soil.

With regard to fungicidal treatment of citrus seeds to prevent storage molds, Ryan (10) found that 75% of Cleopatra mandarin seeds failed to germinate after being immersed for 1 minute in 1% 8-hydroxyquinoline sulfate and stored at 40° F for 2 months in polyethylene sacks, but that seeds dusted with 50% tetramethylthiuram disulfide [thiram] lost none of their germinability during that period. Childs and Hrnciar (3), on the other hand, noted no appreciable effect on the viability of seeds of 34 varieties of citrus and citrus hybrids that had been dipped in 1% 8-hydroxyquinoline sulfate, dried on paper towels, and stored at 35° for 6 to 8 months in moist sawdust or moss in mason jars, with a loose lid to permit aeration.

EXPERIMENTATION

Since it was first necessary to determine what exposures to heat the several seed varieties would tolerate, the following tests were made. Seeds of Glen sour orange, Rangpur lime

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(C. limonia Osbeck), Koethen sweet orange, and Rough lemon were extracted from fruits freshly harvested in Coachella Valley, California and were used immediately without cold storage or chemical treatment. One hundred seeds of each variety were immersed for 10 minutes in water at the temperatures shown in Table 1. They were then planted at once in a peat moss-vermiculite mixture and germination recorded during the following 2 months.

Table 1. Percentage germination of citrus seeds after 10-minute immersion at various temperatures (° F)[a].

Variety	80	120	126	130	135	140
Glen sour orange	94	96	94	95	97	91
Koethen sweet orange	82	96	89	86	79	79
Rangpur lime	93	94	98	92	95	98
Rough lemon	100	99	98	100	100	97

[a]Total germination after 56 days; 100 seeds of each variety were treated and planted.

The results in Table 1 show that under the conditions of the experiment germination of the several varieties of citrus seeds was not impaired by a 10-minute immersion in water at 130° to 140° F. Indications from experiments reported later (Table 5) are that a 10-minute exposure to 130° would be sufficient to disinfect seed against P. parasitica even if oospores were present.

The following results[2] report the effect of heat treatment on germinability of citrus seeds as modified, respectively, by two fungicides and two storage periods. The purpose of a chemical treatment, following the hot-water immersion to destroy Phytophthora spp., is to inhibit the growth of common air-borne molds on the seeds during storage. Seeds of Rough lemon, Cleopatra mandarin, Jochimsen grapefruit, Bessie sweet orange, and Standard sour orange grown in Riverside, California were extracted and refrigerated overnight at 40° to 45° F. They were then washed, immersed for 10 minutes in water at the temperatures shown in Table 2, rinsed in cold water, and immersed for 3 minutes in a 1% solution of 8-hydroxyquinoline sulfate. The seeds were shaken to remove excess fungicide, stored for 2 or 35 days at 40° to 45°, and then tested for germinability in a mixture of peat moss, fine sand, and redwood shavings.

Because seeds of Rough lemon after the 10-minute immersion at 125° F and 3 minutes in the quinoline solution showed very poor germination, tests with this variety were repeated (Table 3). Thiram dust (50% tetramethylthiuram disulfide) was included for comparison. The only known difference in procedure between the preceding tests and these was that in the latter the seeds were not stored in the refrigerator (40° to 45°) overnight, but were heat-treated immediately after extraction and planted after 3 days' storage at 40° to 45°. However, the results of a third test with Rough lemon in which germination was satisfactory indicated that overnight storage was not the cause of the poor germination. Apparently seeds of the four varieties listed in Table 1 tolerated well a 10-minute exposure to 125° and either of the subsequent chemical treatments (3, 10, 11) in spite of the aberrant results obtained with Rough lemon in the first test. To study further the effect of length of storage on heat-chemical treatments, seeds of the several varieties are being held at 40° to 45° for periodic tests of their germinability.

The following tests were designed to secure more evidence on the effectiveness of the hot-water immersion treatment for the destruction of P. parasitica in seeds. Fruits of C. E. S. No. 343 grapefruit, Bessie sweet orange, and Cleopatra mandarin were inoculated with zoospores of P. parasitica and kept until they developed typical Phytophthora brown rot. The seeds were extracted, treated as shown in Table 4, and then inserted in sound apple fruits to permit any living fungus to grow into the apple tissue. Where fungal growth occurred, bits of the decaying apple tissue were transferred to potato-dextrose agar and the causal fungus was identified.

These results are in line with those of previous tests which showed that a 4-minute immersion in water at 120° to 125° F destroyed P. parasitica in citrus seeds (6). The results reported in Table 5 indicate that heat-resistant oospores of the fungus were not present in these seeds.

Mycelium of P. parasitica, bearing abundant oospores taken from a mixture of water infusions of horse manure, sterilized with propylene oxide (4), and apple, was placed in cheese-

[2]Paul W. Moore of W-N Ranch, Thermal, California, participated in tests reported in Table 2.

Table 2. Percentage germination of citrus seeds after heat and subsequent fungicidal treatments[a].

Variety	Storage at 40° to 45° F after treatment (days)	10-minute immersion in water at ° F			
		75	115	125	125
		Dipped in fungicide[b]			No fungicide
Rough lemon	2	96	98	20[c]	91
do.	35	85	90	11[c]	94
Cleopatra mandarin	2	100	100	95	98
do.	35	90	100	88	87
Jochimsen grapefruit	2	100	98	73	86
do.	35	87	78	76	90
Bessie sweet orange	2	73	82	91	93
do.	35	71	80	79	81
Standard sour orange	2	96	88	87	80
do.	35	87	86	88	87

[a]Total germination after 49-day period in a mixture of peat moss, fine sand, and redwood shavings.
[b]Heat-treated seeds rinsed in cold water and immersed in 1% solution of 8-hydroxyquinoline sulfate for 3 minutes.
[c]Low germination was predicted at planting time by a vitality test using a 1% solution of 2,3,5-triphenyltetrazolium chloride (9).

Table 3. Germination of Rough lemon seeds after heat and subsequent chemical treatments.

Temperature of immersion water (° F)	Fungicide[a]	Germination during 39-day period[b] (%)
75	None	97
75	thiram	93
75	8-H	94[c]
115	None	90
115	thiram	96
115	8-H	99
125	None	99
125	thiram	97
125	8-H	97

[a]Thiram was applied as a dust; 8-H indicates a 1% solution of 8-hydroxyquinoline sulfate in which seed were immersed for 3 minutes.
[b]Seed stored 3 days at 40° to 45° F after treatment.
[c]Only 95 seeds in this lot; all other lots had 100.

Table 4. Recovery of *Phytophthora parasitica* from heat-treated citrus seeds.

Variety	10-minute immersion in water at ° F				
	75	115	120	125	130
	Number of seeds with viable fungus				
C. E. S. No. 343 grapefruit (100 seeds for each treatment)[a]	77	0	0	0	0
Bessie sweet orange (40 seeds each treatment)[b]	36	10	0	0	0
Cleopatra mandarin (100 seeds each treatment)[a]	72	3	0	0	0

[a]Four replicate tests, each with 25 seeds for each temperature.
[b]Four replicate tests, each with 10 seeds for each temperature.

Table 5. Growth of P. parasitica following heat treatments in a water bath.

Period of immersion (minutes)	Water bath at °F						
	112	115	118	121	124	130	136
5	+[a]	+	+	+	+	-	-
10	+	+	+	+	±	-	-
15	+	+	+	±	±	-	-
30	+						

[a] + = Fungus survived treatment; - = fungus not recovered; ± = fungus occasionally recovered.

cloth sacks and immersed in water at the temperatures shown in Table 5. Viability of the fungus was determined by inserting the treated material in apple. Bits of apple tissue that showed decay were transferred to potato-dextrose agar for identification of the causal fungus.

CONCLUSIONS

It is assumed that oospores of P. parasitica were not present in the seeds tested, since immersion in water at 120° F for 4 minutes (6) was sufficient to disinfect seeds bearing mycelium and possibly asexual spores of this species and of P. citrophthora. Furthermore, an exposure between 124° for 15 minutes and 130° for 5 minutes was required to inhibit growth from the oospores of P. parasitica. No oospores have been described for P. citrophthora. However, heat-resistant fungus organs possibly may occur in or on naturally infected seeds collected and stored in other ways, and under other conditions seeds of the varieties tested and other varieties may be less tolerant of the higher temperatures used in these studies, that is, 130° to 140° F. It is suggested, therefore, that until other seed collections are tested, an immersion of 125° for 10 minutes be used for seed disinfection, as this treatment assures elimination of P. citrophthora and P. parasitica, without impairing seed germination.

Finally, it should be emphasized that the hot-water treatment of seed obviates only one source of contamination of seedbeds, nurseries, and orchards. Seed treatment can have no sustained, beneficial effect if the soil (7) or the water (8) is infested. Contamination of virgin soil or recontamination of disinfested soil by careless orchard operations can vitiate the beneficial effects of seed, soil, and water treatments to protect citrus against the rootstock-destroying species of Phytophthora.

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FIELD INOCULATIONS OF FORAGE LEGUMES AND TEMPERATURE STUDIES WITH ISOLATES OF SCLEROTINIA TRIFOLIORUM AND SCLEROTINIA SCLEROTIORUM[1]

Raymond A. Cappellini

Summary

Four alfalfa varieties and a red and ladino white clover were artificially inoculated in the field with eight isolates of Sclerotinia trifoliorum obtained from infected alfalfa, red clover, and vetch, and six isolates of S. sclerotiorum obtained from infected lettuce, tomato, peony, and an unidentified species of Draba. Highly significant differences were noted among isolates and hosts and in the isolate x host interaction. Isolates of S. trifoliorum infected all the alfalfas and clovers. Isolates of S. sclerotiorum infected only ladino clover.

In studies at 10°, 15°, 20°, 25°, 30°, and 35°C, the best growth occurred at 15° and 20° and at 20° and 25° for isolates of S. trifoliorum and S. sclerotiorum, respectively. Only the S. sclerotiorum isolates grew at 30°. Isolates of both species failed to grow during 10 days' incubation at 35°.

INTRODUCTION

Root and crown rot caused by Sclerotinia trifoliorum Eriks. can seriously affect the establishment and maintenance of quality forage legumes. In New Jersey, although it can be found in most established alfalfa and clover fields, this disease is especially important on newly seeded fields; up to 100% losses have occurred on such fields (1). In alfalfa stands in the spring following fall seeding, it is not uncommon to find 20 to 30% losses from crown rot.

Held and Haenseler (4) reported that serious outbreaks of lettuce drop (S. sclerotiorum (Lib.) d By.) often occurred in plantings which followed alfalfa or clover. In greenhouse studies, under ideal conditions, they showed that alfalfa, red clover, and other crops were infected by both S. trifoliorum and S. sclerotiorum. Their isolate of S. sclerotiorum killed alfalfa but was only mildly pathogenic to red clover.

In southern New Jersey, the writer has observed the complete destruction, due to crown rot, of two newly seeded alfalfa stands that followed tomatoes, although Sclerotinia had not been reported as a problem on these tomatoes.

The following study was conducted, therefore, to determine the relative pathogenicity of isolates of S. trifoliorum and S. sclerotiorum to various forage legumes under field conditions. The effect of certain temperatures on the growth of these isolates was studied also.

MATERIALS AND METHODS

During the summer of 1957 plants of alfalfa and red clover infected with S. trifoliorum, and lettuce, tomato, and Draba sp. infected with S. sclerotiorum, were collected from eight New Jersey counties. Twelve isolates of these species were obtained, and subsequently maintained, by sclerotial plantings on Difco potato-dextrose agar (4). Inoculum was prepared by infesting sterile wheat grain (5). Experimental plots measured 9 x 12 feet and were arranged as a randomized block with four replications. Atlantic alfalfa was band-seeded, and fertilized in mid-August 1957, as recommended for New Jersey. Approximately 3 months later the plants in each plot were inoculated with each isolate at the rate of 1 gram of inoculum per square foot of plot (3). Observations on stand were made prior to dormancy and disease data were taken in April 1958, after spring re-growth, from the central 100 inches in each "band" in each plot.

When a similar experiment was performed in the fall of 1958 the same procedures were followed. All isolates but one from lettuce (#14) were tested in addition to one each from vetch (S. trifoliorum) and peony (S. sclerotiorum). The alfalfa varieties Atlantic, Dupuit, Northwestern and Vernal, and Penscott red and ladino white clover were planted. The Atlantic alfalfa plots were discarded because of a poor stand attributed to a poor lot of seed.

In the temperature study, 11 isolates were grown in triplicate on PDA and incubated at 10°, 15°, 20°, 25°, 30°, and 35°C. A 5-mm inoculum-disk was planted on the agar at the edge of a 90-mm Petri dish, and radial growth was measured in mm.

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RESULTS AND DISCUSSION

Field Inoculation Studies: The results of the study with Atlantic alfalfa showed wide differences in the pathogenicity of the S. trifoliorum isolates. Percentage of infected or dead plants ranged from 0 to 64 (Table 1). The lack of infection with the S. sclerotiorum isolates was surprising and in direct contrast with the results obtained in the greenhouse by Held and Haenseler (4). In the 1958 test, not only did the isolates differ significantly in pathogenicity but significant differences were noted among hosts and in the host x isolate interaction (Table 1).

Table 1. A comparison of field pathogenicity of isolates of S. trifoliorum and S. sclerotiorum on alfalfa and clover.

	% crown rot[a] obtained with:														
	S. trifoliorum isolates[b]								S. sclerotiorum isolates[b]						
Hosts	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Control
1957 Test															
Atlantic	57	18	61	0	20	64	59	-	-	0	0	0	0	0	0
F value - isolates = 131.24**															
1958 Test															
Northwestern	0	18	55	16	44	33	57	31	0	0	0	0	0	-	0
Vernal	0	15	9	14	53	41	32	10	0	0	0	0	0	-	0
Dupuit	9	13	53	16	51	52	49	24	0	0	0	0	0	-	0
Penscott	0	19	50	53	63	64	55	52	0	0	0	0	0	-	0
Ladino	0	51	55	33	51	64	44	53	31	8	11	0	24	-	0
F values - isolates = 61.03**; hosts = 54.14**; isolates x hosts = 5.31**															

**Significant at the 1% level.

[a] Calculated, after spring regrowth, as inches of infected or dead plants by Sclerotinia in 100 inches of row measured. Average of four replicates.

[b] Isolates obtained from:

1. alfalfa
2. alfalfa
3. red clover
4. alfalfa
5. alfalfa
6. red clover
7. alfalfa
8. vetch
9. peony
10. lettuce
11. tomato
12. tomato
13. Draba sp.
14. lettuce

Table 2. A comparison of the average growth of seven isolates of S. trifoliorum and four isolates of S. sclerotiorum on Difco PDA at various temperatures.

	Radial growth in mm after 5 days[a]					
	Temperature (°C)					
Species	10	15	20	25	30	35
S. trifoliorum	32	53	53	21	0	0
S. sclerotiorum	41	68	86	88	26	0

[a] Average of three replicates.

The S. sclerotiorum isolates produced no infection on alfalfa and red clover, and only 0 to 31% on ladino white clover. The apparent lack of pathogenicity of these S. sclerotiorum isolates to the alfalfas and red clover under these conditions was not investigated. One might speculate that environmental conditions are inherently related to the expression of pathogenesis between these isolates and certain "hosts."

Kreitlow and Sprague (6) reported marked differences in pathogenicity to ladino clover among monoascospore isolates of S. trifoliorum. The results reported herein essentially agree with their observations and indicate the possible existence of natural pathogenic strains within this species. The data also seem to indicate a relatively feeble relationship between outbreaks of crown rot in the forage legumes and certain crops susceptible to S. sclerotiorum that are followed in the rotation.

Temperature Studies: The results of these studies are in general agreement with several authors (2, 6, 7, 9). The main point of interest in these studies is that, while morphological differences between S. trifoliorum and S. sclerotiorum are difficult to assess (8), at least certain

isolates within these two species exhibit vast physiological differences. The temperatures showing the best growth for the S. trifoliorum isolates were at 15° and 20° C; for the S. sclerotiorum isolates, at 20° and 25° (Table 2). The S. trifoliorum isolates failed to grow at 30°, but the S. sclerotiorum isolates averaged 26- and 58-mm growth after 5 and 10 days, respectively. Both species failed to grow at 35°. The S. trifoliorum isolates held at 30° for 10 days were placed at 20°; within 2 weeks they had completely grown over the dish and produced normal-appearing, viable sclerotia. The plates held at 35° were discarded after 10 days.

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A SPADIX ROT OF ANTHURIUM IN HAWAII[1]

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Abstract

Colletotrichum gloeosporioides was found to be the causal organism of anthurium spadix rot, a previously unreported disease. The disease, principally affecting the rudimentary perianth, develops rapidly under favorable conditions of high temperatures and high humidity. Maneb, dodine, and 2,4-dichloro-6-(o-chloroaniline)-s-triazine as protectant fungicides and hot-water treatment at 46°, 47°, and 48° C for 15 and 30 minutes gave excellent control of the disease.

INTRODUCTION AND LITERATURE REVIEW

A spadix rot of anthurium (Anthurium andraeanum), observed previously but never known to be widespread, became epidemic during the early fall of 1959 in the high-rainfall areas on the island of Hawaii. Considerable loss due to the disease was incurred in the field and in transit, and there was deep concern in anticipation of further loss during the ensuing rainy winter months.

Colletotrichum anthurii (All.) Neergaard, which is considered to be synonymous with C. gloeosporioides by von Arx (2), has been reported as occurring on the foliage of Anthurium scherzerianum in the Netherlands (5). Fischer (4) and Wahl (7) reported a circular brown leaf spot occurring on leaves of A. scherzerianum caused by Gloeosporium minutum in Austria. G. minutum is not among the 600 names which von Arx (2) has reduced to the synonymy of C. gloeosporioides. Glomerella cincta (Ston.) Spauld. & Schrenk has been reported to occur on an undetermined species of Anthurium by Seymour (6).

Ciferri (3) reported that floral spathes of A. scherzerianum were occasionally infected by weakly parasitic Fusoma calidariorum. Weiss and O'Brien (8) did not report any anthurium disease in their index of plant diseases.

C. gloeosporioides Penz. and a species of Cladosporium were constantly associated with the disease, but inoculations showed that only C. gloeosporioides was capable of infection. Within 2 to 4 days after inoculation, the rot starts as tiny black spots on the scale-like components of the perianth, and occasionally on the exposed ovarian walls. During conditions of high humidity the spots enlarge, become watery, turn brown, and soon these parts are completely affected (Fig. 1); during the early stages these infection sites remain isolated and rarely

FIGURE 1. Typical spadix rot symptoms in the early stages. Healthy spadix on the left.

[1] Published with the approval of the Director of the Hawaii Agricultural Experiment Station as Technical Paper No. 502.

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serve as centers of further spread from which adjacent flowers are directly infected. Thus, the shape and size of the spots (sharply angular and delimited) are determined by the surface of the individual perianthal component. As the disease progresses, pinkish spore masses of the causal organism become evident, and the affected tissue dries and pulls away from the unaffected portions of the spadix. If the infection is severe, accompanied by high humidity and temperatures, the rot extends into the peduncle and causes the whole spadix to rot and turn brown within a week.

The foliage leaves and spathes are not affected and all inoculation attempts have shown no signs of disease.

Since no report could be found which definitely cites an inflorescence disease of anthurium to be caused by C. gloeosporioides or any of its numerous synonyms, it is believed that this is the first report of anthurium spadix rot.

MATERIALS AND METHODS

Cut flowers of the varieties Kaumana and Suyehiro White (A. andraeanum) were used for most of the tests. Flowering plants of A. scherzerianum[3], and papaya fruit (Carica papaya) treated with hot water at 48° C for 20 minutes, according to Akamine and Arisumi (1), were used in the inoculation studies.

Several isolations of C. gloeosporioides were made from anthurium spadix rot, papaya anthracnose, mango anthracnose, and leaves of passion fruit. C. lindemuthianum and C. lagenarium (C. orbiculare) considered by von Arx (2) as being specialized forms of C. gloeosporioides, were also included in the tests.

The following chemicals, at the respective dosages, were tested for their ability to control spadix rot: nystatin (100 ppm + 0.3% dimethyl sulfoxide), cycloheximide (Acti-dione) (5 ppm), zinc ethylene bisdithiocarbamate (zineb) (65% WP, 2 pounds/100 gallons), manganese ethylene bisdithiocarbamate (maneb) (70% WP, 2 pounds/100 gallons), N-trichloromethylmercapto-4-cyclohexene-1, 2-dicarboximide (captan) (50% WP, 2 pounds/100 gallons), tetramethylthiuram disulfide (thiram) (75% WP, 2 pounds/100 gallons), n-dodecylguanidine acetate (dodine[4]) (65% WP, 1 pound/100 gallons), and 2, 4-dichloro-6-(o-chloroaniline)-s-triazine[5] (50% WP, 1 pound/100 gallons). Wetting Agent 60-L (60% solution of an alkyl aryl sulfonate) was added to all of the chemical treatments at the rate of 1:2000. The spadices were allowed to dry before inoculations were made.

The inoculum was prepared by washing off spores from 7-day-old cultures grown on vegetable juice agar[6]. The spore suspensions (200,000-400,000 conidia per ml) were applied with a paint sprayer and the inoculated spadices were allowed to dry before being placed in moist chambers.

Temperatures of 46°, 47°, and 48° C, each for 15-, 30-, and 60-minute periods, as suggested by the success of Akamine and Arisumi (1), were used for the hot-water treatments. Spadices were inoculated as just described, incubated for 24 hours, then placed in the water baths; immediately after the treatments, the spadices were plunged into cold water, then returned to moist chambers to complete the incubation.

RESULTS

All of the C. gloeosporioides isolates from anthurium and one of the isolates from papaya obtained from Puna, East Hawaii, were pathogenic to anthurium spadix; the symptoms were identical with all isolates. The other isolates, including two papaya isolates from Oahu, were not capable of inciting the disease. Foliage leaves and spathes were unaffected.

The isolate from papaya, after recovery from anthurium spadix, was still capable of causing papaya anthracnose. All of the C. gloeosporioides isolates from anthurium spadix rot were capable of causing papaya anthracnose, and upon reisolation from papaya were still able to cause anthurium spadix rot.

The youngest of three spadices, none of which were fully expanded, of A. scherzerianum was affected by spadix rot at the apical end, which is comprised of the least mature flowers. The foliage leaves and spathes were unaffected, indicating that the spadix-rot organism is dis-

[3]Anthuriums were generously supplied by Dr. H. Kamemoto of the Horticulture Department, Hawaii Agricultural Experiment Station.

[4]Cyprex 65-W, supplied by American Cyanamid Co.

[5]Dyrene, supplied by Chemagro Corporation.

[6]Medium composed of 10% V-8 juice, a proprietary vegetable juice product, 0.2% $CaCO_3$ and 2% agar.

tinct from the European Colletotrichum.

Maneb, dodine, and Dyrene gave excellent control of the disease . Thiram and captan were intermediate in disease control and the others were no better than the untreated controls.

Maneb was further tested under an environment simulating field conditions. Spadices of Kaumana and Suyehiro White, grown in a lath house, were sprayed with maneb; after 7 days, they were inoculated and incubated as previously described. Control of spadix rot by maneb under these conditions was excellent.

Sixty-minute treatments of all three temperatures scalded the spathes which turned brown, then black within 24 hours; the spadices also started to dry out after 4 days. Control was excellent with all other hot-water treatments, although a gradient was observed in which the smallest and least number of spots occurred at the highest temperature exposed for 30 minutes.

Keeping quality tests were not conducted for the chemical or hot-water treatments, but all the spadices of the effective treatments remained in excellent condition for 2 weeks or more, whereas the infected controls deteriorated in less than a week. No rot developed in the uninoculated controls of any of the treatments.

DISCUSSION AND CONCLUSIONS

Von Arx (2) in his comprehensive work on the species concept of Colletotrichum reduced some 600 names to the synonym of C. gloeosporioides. However, under this name he retained several names of specialized forms on certain hosts. Among these is C. musae, characterized as being rapid growing, infecting various tropical fruits, and possessing glabrous acervuli.

In our experience with numerous isolates of C. gloeosporioides from many tropical crops, we have found that most of them are unspecialized and that their acervuli may be glabrous or setose. Isolates of the anthurium spadix rot organism also possessed glabrous or setose acervuli and have shown some degree of specialization, in that, with a single exception, none of the other anthracnose-causing organisms tested was able to infect anthurium spadix.

The anthurium spadix rot organism has been placed under the name, C. gloeosporioides, in view of these considerations and also for the following reasons:

1. Culturally, the isolates of various C. gloeosporioides have been highly variable on different media and environmental conditions.
2. Morphologically, however, the isolates have been quite uniform, and they all fell within the limits of the species.
3. Although the degree of host specialization is high, ability to cross-inoculate with a papaya isolate was demonstrated.
4. To set aside a name, C. musae, to indicate specialization on host species representing widely divergent families such as Musaceae, Araceae, Caricaceae, Anacardiaceae, may be confusing.

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LATE FALL APPLICATION OF FUMIGANTS FOR THE CONTROL OF SUGAR BEET NEMATODES, CERTAIN SOIL FUNGI, AND WEEDS[1]

Jack Altman[2] and B. J. Fitzgerald[3]

Abstract

High concentrations of D-D (1,2-dichloropropane -- 1,3-dichloropropene) were injected into Heterodera schachtii-infested soil in November 1958, to a depth of 7 inches in-the-row. At the same time 5-inch ridges were made over the injection site resulting in a fumigant placement at a 12-inch depth. In the spring of 1959 the top layer of the ridges was removed and beets were planted on freshly exposed soil. Growth of the plants in treated plots in July was four times that of beets in the non-treated plots. Yields at harvest from treated plots were twice the yields from non-treated plots.

INTRODUCTION

The application of nematocides to the soil is normally recommended as a spring preplant application at soil temperatures of 55° F or higher. Since most of the fumigants are volatile liquids whose toxic effects are usually exerted as a gas that permeates the soil, killing soil microorganisms on contact, the higher soil temperatures have been required to effect the liquid to gas conversion and the diffusion of the gas through the soil. Diffusion of the nematocide is also generally reduced under high soil moisture conditions (2).

In Colorado, heavy clay soils are often too wet and too cold to fumigate early in the spring, but waiting until soil temperatures are sufficiently high (55° F or higher) to fumigate may delay planting 2 or 3 weeks. Sugar beet growers in northeastern Colorado are usually reluctant to apply any material that will result in a delay in spring planting.

With the commercial nematocides presently available, toxicity probably depends upon volatilization of the chemical in a short period of time, at concentrations sufficiently high to kill nematodes and other soil microorganisms. Alternatively, however, instead of the release of lethal concentrations of nematocide in a short period of time (as with spring fumigation), if low concentrations of the compound are placed in contact with the nematode over a prolonged time period (3 to 5 months), they may be equally effective in controlling the nematode. Also, when fumigating is done under environmental conditions where volatility is reduced -- particularly when soil temperatures are low -- a simple compaction with a cultipacker may effectively seal the nematocide in the soils.

MATERIALS AND METHODS

Two experiments, one a 4 x 4 latin square designated as test No. 1 and the second a 2 x 5 randomized block referred to hereafter as test No. 2, were set up in late fall of 1958 to test the effect of D-D, injected as a deep chisel treatment, on the control of soil microorganisms and weeds. The 4 x 4 latin square consisted of the following treatment in 100-foot-long plots: 1)Control (non-treated), 2)Weed, 3)D-D and weeds, 4)D-D. The "weed" treatment consisted of sowing a composite mixture of green foxtail, hog-millet, pigweed, dropseed, watergrass, and rye on the designated plots. D-D was used at 14 gallons per acre. The 2 x 5 randomized block test was set up in alternating 400-foot 4-row plots consisting of control and D-D treated plots. No weed treatment was included in these plots.

The fumigant was injected with a tractor-drawn set of "chisels" to a 7-inch depth, into soils previously prepared as seed beds. A four-chisel apparatus made by the senior author was mounted on the front draw bar of a Model M-IH Tractor. Fumigant from a supply tank was pumped by power-take-off pump into each "chisel applicator." T-jet spray tips, placed in the line secured to back of the chisel, served as metering devices. Chisels were set 22 inches apart, at sufficient depth to permit placing of the fumigant 7 inches deep. Five small ditching shovels were attached to the rear draw bar of the tractor 11 inches to either side of the front chisels. During operation these shovels were lowered, to make four ridges or beds 5 in-

[1]Published with the approval of the Director of Colorado Agricultural Experiment Station, Fort Collins, Colorado as Scientific Series Paper No. 684.

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ches high. When the front chisels and rear shoes were in position the fumigant was placed at a 12-inch depth in the row.

Composite soil samples in each plot were taken at a 4 to 6-inch depth on July 8 and August 24, 1959. Half of each sample was washed and examined for the presence of the sugar beet nematode and half was examined for presence of soil-inhabiting fungi by means of the Mueller-Durrell Sampling tubes (3).

DISCUSSION AND CONCLUSIONS

One of the objectives of the tests was to point out the relationship between nematode counts and yield. From the data in Tables 1 and 2, D-D apparently did reduce the population of *Heterodera schachtii* larvae, and the reduction may account for the yield difference recorded in Tables 3 and 4. However, in the previous year's work with spring fumigated plots, this reduction in nematode larvae was not a reliable measurement for determining the effectiveness of nematocides. At certain sampling dates nematode populations in the non-fumigated plots were also reduced. After hatching from cysts the nematodes migrate toward a suitable host food source. However, few larvae are successful in locating a host plant and as a consequence many of them die; hence there is a tremendous reduction in the nematode population in both treated and non-treated plots.

The harvest data in Tables 3 and 4 indicate that D-D may have had some effect in altering the soil rhizosphere or microbial population in favor of the host, resulting in a better top growth and root development in treated plots. Yields from control plots in both test areas were low, owing partly to late planting and the presence of nematodes or other debilitating microorganisms, and to a large extent to the improper thinning which left numerous "doubles" and "triples" in the field. Since planting, thinning, and irrigating in the test were carried out by the farm on which the test was located, the unusual number of "double" and "triple" beet roots was not detected until harvest. Sugar beets on the same ground the previous year yielded 6 tons or less of roots to the acre.

All factors considered, the harvest data in Tables 3 and 4 indicate that in both tests D-D almost doubled the yield over that of the controls (Fig. 1).

FIGURE 1. Results of harvest of 100 feet of row from control (right) and treated (left) plots.

In addition to increasing yield, application of the fumigant in late fall prior to planting seemed to have some effect in reducing the weed population. Table 5 contains a count of weeds in the 4 x 4 latin square test 3 months after thinning, with the plots remaining undisturbed until these data were tabulated. Observations in late August indicated that bindweed, grasses, and pigweed from natural weed populations were reduced extensively in the 2 x 5 randomized block test (400-foot rows). The weed reduction may have been due in part to a direct effect of the chemical on the weed seed and in part to shading from more vigorous growth of beet foliage in the D-D plots, causing a reduction in weed seed germination and weed growth.

In regard to effect of the fumigant on soil fungi, extensive isolations of fungi trapped in "soil tubes" incubated in containers of soil from individual plots indicated a tendency toward a

Table 1. Sugar beet nematode population densities per quart of soil in test No. 1[a].

Treatment	July 8, 1959 Empty cysts	July 8, 1959 Full cysts	July 8, 1959 Larvae	August 24, 1959 Empty cysts	August 24, 1959 Full cysts	August 24, 1959 Larvae
Control	41.4	5.8	16	28	16.4	66
Weed	32.5	5.2	59	28	7.4	64
D-D + weed	48.8	3	17	22.2	8.8	26
D-D 14 gallons/acre	33.4	3.6	6	28.8	11.2	22

Plot length 100 feet.

[a]This test was located at Windsor, Colorado and fumigated November 4, 1958. Data are based on four plot averages.

Table 2. Sugar beet nematode population densities per quart of soil in test No. 2[a], July 8, 1959.

Treatment	Empty cysts	Full cysts	Larvae
Control	79.8	9.6	251
D-D 14 gallons/acre	73.4	9.2	8

Plot length 400 feet.

[a]Fumigated November 4, 1958. Data are based on five plot averages.

Table 3. Summary of 1959 harvest results of sugar beets from fumigated plots in test No. 1.

Treatment	Number roots per plot[a]	Tons of roots/acre[b]	% sucrose[c]	Gross tons sucrose/acre
Control	183	5.3	14.34	.76
Weed	150	4.1	14.41	.59
D-D + weed	281	10.8	13.13	1.42
D-D	238	9.1	13.82	1.26
LSD 5%		2.33		.50
LSD 1%		2.91		.72

[a]Plot length 100 feet.

[b]Based on the population of living plants from the center two rows of each plot (190 feet of row harvested per plot). All data are based on four plot averages.

[c]Based on duplicate analysis of each of three composite samples per plot. Each composite sample consisted of not less than 20 roots where possible.

Table 4. Summary of 1959 harvest results of sugar beets from fumigated plots in test No. 2.

Treatment	Number roots per plot[a]	Tons of roots/acre[b]	% sucrose[c]	Gross tons sucrose/acre
1. Control	487.0	6.17	15.03	.93
2. D-D	661.4	11.83	14.48	1.71
Calculated "t"		8	4.81	8.62

"T" test for significance for paired data: 5% = 2.78; 1% = 4.604.

[a]Plot length 400 feet.

[b]Based on the population of living plants from the center two rows of four 50-foot sections of each plot. (400 feet of row harvested per plot). Data are based on five plot averages.

[c]Based on duplicate analysis of eight composite samples per plot.

Table 5. Post thinning weed count from test No. 1[a], September 1, 1959.

Treatment	Broad leaf weeds	Grasses
Control	34.75	27.75
D-D + weed	7	16.75
Weed	26.75	34.75
D-D	10	14.25

[a]Based on entire population of weeds remaining in the center two rows of each plot after prior weeding and thinning in June 1959. The data represent four plot averages for 200 feet of row.

reduced frequency of Fusarium invasion of the soil tubes from the D-D plots (3). In some instances Mucor sp. and Pythium sp. seemed to compete and replace the Fusarium. In the control plots in both tests Fusarium was the predominant fungus trapped by the "soil tubes," since it was recorded from more than 90% of the isolates made from the tubes. In a field with high incidence of Rhizoctonia fumigated the previous year on another farm and replanted to beets, stand counts late in the season in areas that had been fumigated indicated less Rhizoctonia and a greater number of surviving beets than in non-fumigated areas.

The method and time of application of the D-D is somewhat unique in that instead of applying when soil temperatures were high, 55° to 70° F (or suitable for fumigation as recommended by the manufacturer), the fumigant was applied when the soil temperatures were low (40°). Under these conditions the gas probably vaporized less through the soil and consequently prolonged the effect (perhaps 5 months or longer) of the fumigant (1). Observable beet top growth in fumigated plots was at least three times (sometimes four times) that of top growth of beets in non-fumigated plots throughout the entire 1959 growing season. There seemed to be very little lateral movement of the fumigant to control plots, since the observable increase in top growth and general increased vigor was limited to the fumigated beets and these plots were very sharply delimited from the control (non-treated) plots. The fumigant was applied after all other farm operations for the year had been completed and a 2-week delay in planting in the spring, as recommended after normal fumigation, was thus avoided. A spring fumigation test was set up adjacent to the fall test. Comparisons throughout the growing season showed a more vigorous growth and stand in the late fall fumigated plots than in the spring fumigated plots. One other point is that the application of the fumigant was relatively simple and that placement of the fumigant in the row permitted the use of economical and practical gallonage.

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RESISTANCE TO PHYTOPHTHORA ROOT ROT IN PEPPER

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In 1941 Tompkins and Tucker (5) reported a destructive root rot of pepper and pumpkin in the San Joaquin Valley of California. They also cited several reports of this disease, caused by Phytophthora capsici Leonian, in pepper in other States and in several widely separated countries. A similar disease has recently occurred sporadically in California pepper fields. Damage has occasionally been severe, and usually most prevalent in fields that received excessive irrigation.

A destructive root rot of pepper near Patterson (San Joaquin Valley) was called to our attention in 1957. Isolations from plants from the field yielded cultures of Phytophthora sp. that inoculated to nearly mature California Wonder pepper plants in the greenhouse caused a rapid wilting and killing very similar to the disease syndrome in the field. The fungus was identified as P. capsici.

This paper reports the finding of sources of resistance to this disease in lines of pepper compatible with Capsicum annuum.

METHODS

Dr. Paul Smith, Department of Vegetable Crops, University of California, Davis, furnished 613 seed samples of various pepper varieties and plant introductions, all genetically compatible with Capsicum annuum. Screening for tolerance or resistance to Phytophthora was done as follows: All seeds were heat-treated in water for 30 minutes at 52° C to eliminate seed-borne Rhizoctonia solani (1). (Before this procedure was adopted, seedling damping-off seriously interfered with interpretation of results.) Seed were planted in 1:1 sand-peat in flats with additional nutrients (Fertilizer I) as described by Baker (2). Seven rows, each 2 1/2 inches apart, were seeded in each flat. One row in each flat was seeded with a susceptible check (California Wonder); each of the other six rows was seeded with one test line. A mycelial suspension of Phytophthora was made by blending mycelial mats from 10-day-old test-tube (200 x 25mm) slants with distilled water in proportion of two mats per 200 ml of water. Plants in the 8- to 10-leaf stage were inoculated by pouring 25 ml of the mycelial suspension into furrows, 1/2 inch deep, between the rows. Surviving plants were counted at 30 days. Of the 613 lines tested, 13 appeared more resistant to Phytophthora root rot than susceptible California Wonder which was usually killed completely within 5 days of inoculation.

The 13 lines were retested with zoospore suspensions. Zoospores were produced by a modification of the procedure described by Gooding and Lucas (4). The fungus was grown on oatmeal agar in Petri plates. After 20 days' growth at 25° C the mycelial mats were stripped away from the medium, cut into strips 1/2 inch wide, placed in sterile Petri plates, flooded momentarily with 0.05% KNO_3 solution, and incubated 5 to 7 days at 24° to 26° C. Abundant mature sporangia were produced on the surface of the mats. Zoospore release was induced by flooding the mats with water and chilling for 20 minutes at 4° C. The number of zoospores per ml of the suspension was estimated by counting a sample in a haemocytometer, and the content was adjusted with water to about 5000 zoospores per ml. Furrows were made between the rows as previously described, and 25 ml of the zoospore suspension was poured into each furrow. Inoculations were made in the late afternoon or at night, when greenhouse air temperatures were about 55° to 65° F. The flats were watered liberally until the test was completed. The number of plants showing definite symptoms was recorded at 5-day intervals for 30 days (Table 1).

Five of the PI selections, 187331, 123469, 201232, 188476, and 201234, had relatively high resistance, with percentage of survivors greatest in PI 201234. The variety Jalapeno is decidedly more resistant than California Wonder, but much less so than the five PI selections.

All plants from seed obtained from surviving plants of PI 201234 survived when tested by the above procedure.

A culture of P. capsici (obtained from R. E. Stall, Indian River Field Laboratory, Fort Pierce, Florida) was used in testing plants of PI 201234 and California Wonder. The PI selection was very resistant, whereas all plants of California Wonder were killed. Señora Consuelo Bazán de Segura, of Peru, tested PI selections 201232 and 123469, and found them resistant to Peruvian isolates of P. citrophthora (Smith & Smith) Leonian, the reported cause of Phytophthora root rot of pepper in Peru (3). (Information concerning results of the tests received in personal communication to Dr. Paul Smith who had sent the PI selections to Senora Bazán de Segura.)

Table 1. Resistance of 14 pepper varieties and PI selections to inoculation with a zoospore suspension of Phytophthora capsici.

Seed sample	Percentage of plants diseased at 5-day intervals						Total plants tested
	5	10	15	20	25	30	
California Wonder	100						320
Chile Pequin	91	100					182
201235	90	95	97	100			40
159233	66	98	100				50
173774	90	97	97	97	98	100	105
Poblano	68	71	84	93	100		31
159255	17	63	83	89	91	98	134
188473	36	62	77	87	92	94	56
Jalapeno	37	50	58	63	69	70	110
187331	18	28	31	32	34	38	141
123469	12	20	25	27	28	29	143
201232	4	10	12	14	14	14	91
188476	2	7	7	7	13	15	59
201234	3	4	4	6	7	7	95

Inheritance of the resistance is under study and attempts will be made to incorporate the resistance into bell pepper types similar to California Wonder.

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A SPRAY METHOD FOR INOCULATING SUGARCANE SEEDLINGS WITH THE MOSAIC VIRUS[1]

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Abstract

A spray method for inoculating sugarcane seedlings with the mosaic virus at Meridian, Mississippi has been very promising in limited tests. An inoculum containing fine sand was sprayed onto the seedlings from an atomizer operating at 120 to 150 pounds per square inch. The spray technique is very rapid, has given higher infection rates than the standard rubbing method, and may prove suitable for inoculating sugarcane seedlings in flats before transplanting.

The method currently in general use for artificially inoculating young sugarcane seedlings with the mosaic virus was introduced by Bain (1) in 1944. It involves rubbing each seedling by hand with an inoculum containing an abrasive, usually sand or carborundum powder. The procedure is faster than the Matz method (2), which it largely replaced, but is laborious when large seedling populations are screened for resistance to mosaic. Approximately one million seedlings are inoculated annually in the United States, and the number is expected to increase.

Approximately 14,000 sugarcane seedlings are inoculated each year at the U. S. Sugar Crops Field Station, Meridian, Mississippi. Seed are germinated in the winter in greenhouse flats. The young seedlings are space-transplanted (2 x 2 inches) into other flats where they are inoculated with the mosaic virus as soon as they become well established. Seedlings that develop mosaic symptoms are discarded; healthy seedlings are set into the field in the spring. Normally the seedlings are inoculated only once; the overall infection rate ranges from 20 to 25%. However, repeated inoculation of approximately 800 seedlings, representing 29 crosses, resulted in an infection rate of 50% in 1957 and 1958. One inoculation by the Bain method apparently permits many susceptible seedlings to escape infection. The time required for repeated inoculation by the Bain method prohibits the use of that procedure for routine screening in the breeding program.

In an attempt to develop a more rapid method of inoculation, an experiment designed to test a spray technique was initiated on March 10, 1960. (Richards and Munger (3) successfully used a spray method for inoculating beans and cucumbers with viruses in 1944.)

Expressed sap of the sugarcane variety Co. 290 infected with Strain B of the sugarcane mosaic virus was diluted with equal parts of tap water. To this mixture was added about 3 g of 240-mesh silica per 100 ml of liquid. Inoculum prepared in this manner was sprayed onto sugarcane seedlings with the apparatus shown in Figure 1. The sprayer consists of an atomizer fitted with a by-pass air line running into the inoculum. Air from the by-pass line provides agitation for keeping the silica in suspension. The atomizer was operated at an air pressure of 120 to 150 pounds per square inch.

Sugarcane seedlings in one flat from each of six crosses were inoculated by the spray method. Seedlings of each cross in 2 to 10 additional flats were inoculated by the Bain method using 80 to 100 mesh silica as the abrasive. Each flat in the test contained approximately 54 seedlings. Final mosaic readings were made 1 month after inoculation.

Results of this test are presented in Table 1. The percentage infection from spray inoculation was in the general range of that which has been obtained by repeated inoculation with the Bain method in other tests. The spray method gave a substantially higher infection rate than the Bain method in the progeny of each cross.

In the test just described, no attempt was made to determine the time required to inoculate a flat of seedlings by the spray method. Since no sugarcane seedlings were available for further testing, sorgo seedlings were used in a test to determine how thoroughly plants must be sprayed to give an adequate rate of infection.

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[1]Cooperative investigations between the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Mississippi Agricultural Experiment Station.
[2]Plant Pathologist, Agricultural Research Service, United States Department of Agriculture, Meridian, Mississippi.

FIGURE 1. Apparatus for spray inoculating sugarcane seedlings with mosaic virus. (A) air inlet; (B) by-pass air control valve; (C) by-pass air line; (D) air inlet shut-off valve.

Table 1. Comparison of two methods of inoculating sugarcane seedlings with the mosaic virus.

Cross	Bain method		Spray method	
	Number of seedlings	Mosaic-infected seedlings (%)	Number of seedlings	Mosaic-infected seedlings (%)
C.P. 43-18 x C.P. 50-38	161	11.2	54	77.8
C.P. 44-155 x C.P. 52-43	432	12.3	53	34.0
Cl. 41-142 x C.P. 56-12	262	22.5	53	34.0
C.P. 55-56 x F. 46-345	107	19.6	54	51.9
C.P. 31-511 x F. 46-345	155	12.3	53	71.7
C.P. 48-117 x L. 54-4	158	24.1	54	83.3
Total seedlings	1275		321	
Average % infected seedlings		17.0		58.8

Five flats of Sart sorgo seedlings (54 seedlings per flat), one flat for each treatment, were used in the test. All plants were inoculated in the 3-leaf stage. Plants in one flat were inoculated by the Bain method, whereas, those in the other four flats were inoculated by spraying 2, 4, 6, and 8 seconds, respectively. The number of seconds corresponds also to the number of times the spray was swept the length of the flat of seedlings. For example, in the case of the flat sprayed 4 seconds, the spray was swept from one end of the flat to the other four times, with each sweep requiring about 1 second. Mosaic infection was determined 2 weeks after inoculation.

The percentages of infection for spraying were 83, 98, 100, and 100, respectively, for the 2-, 4-, 6-, and 8-second treatments. The Bain method (check) gave 87% infection, about the same rate as the shortest spraying time (2 seconds).

The atomizer used in these tests delivers about 2.2 ml of inoculum per second at 120 to 150 pounds per square inch air pressure; thus, the quantity of inoculum required by the spray method

is not excessive. An atomizer designed to permit independent regulation of air and liquid output would probably make possible a saving in inoculum. Since a flat containing 500 seedlings can be covered by the spray as quickly as a flat containing 50 seedlings, the quantity of inoculum required per plant inoculated is determined largely by the stand density of the plants.

Stand density is greatest in flats before the seedlings are space-transplanted. Apart from the conserving of inoculum, inoculation of such flats has the more important advantage of obviating much of the wasted effort of transplanting susceptible seedlings. Sugarcane seedlings are probably susceptible to mosaic as soon as they emerge, but they cannot be inoculated by the Bain method until they are large enough to withstand the mechanical injury of rubbing. In a test not previously mentioned, 100% infection of 271 newly emerged sorgo seedlings was obtained by the spray method without noticeable mechanical injury. It seems likely that sugarcane seedlings also could be inoculated soon after emergence. If so, most susceptible plants probably could be eliminated before transplanting.

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PINK ROOT DISEASE OF ONIONS IN MINAS GERAIS, BRAZIL

Geraldo M. Chaves[1] and Homer T. Erickson[2]

Pink root of onions (Allium cepa), caused by Pyrenochaeta terrestris (Hans.) Gorenz, J. C. Walker & Larson, was found in the research plots of the Escola Superior de Agricultura, Viçosa, Minas Gerais, in September 1959. This note appears to be the first published record of the fungus in Brazil.

In September of 1960 the authors surveyed a number of commercial fields in the Ubá, Rodeiro, Guidoval region of Minas Gerais. Results indicate that the disease occurs extensively in the area, where it also attacks garlic (Allium sativum). Despite its frequency, pink root does not appear to be a serious disease in this part of Brazil.

The fungus has been obtained in pure culture and inoculation experiments will be initiated next year.

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CEPHALOSPORIUM STRIPE ON SMALL GRAINS IN ILLINOIS

J. W. Gerdemann, and R. O. Weibel

Cephalosporium stripe caused by Cephalosporium gramineum Nisikado & Ikata was first found on wheat in the United States by Bruehl (1, 2, 3) in the State of Washington. Since then it has been reported on wheat in New York (5) and Montana (4).

During the spring of 1960 Cephalosporium stripe was found on winter wheat in five fields in east-central Illinois. At one location it was also found on barley, oats and rye.

Apparently this disease has been present for a number of years at the Newton Soils Experiment Field in a field that has been in continuous wheat. Considerable losses have been experienced in this field during the past 3 years. The disease was also destructive on wheat in small areas in one other field where wheat had followed wheat. In all other fields it was present in only trace amounts and was not causing any appreciable loss of grain.

The symptoms of the disease were very similar to those described by Bruehl. On wheat, barley and rye the disease was characterized by long yellow to white stripes on the leaves and leaf sheaths. On oats the stripes tended to be reddish. Brown vascular bundles were closely associated with stripes. Severely infected plants were stunted and produced small white heads that were either completely sterile or poorly filled with shrunken grain.

The fungus was readily isolated by plating bits of leaf or leaf sheath tissue containing a discolored vascular bundle on potato-dextrose agar. All isolates were very similar in cultural appearance, growth rate, and morphology to those described by Bruehl.

Bruehl (3) and Tyler and Dickens (5) reported barley, rye and oats susceptible when artificially inoculated. However, this apparently is the first report of this disease occurring naturally in the field on these crops in the United States.

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BOOK REVIEWS

"THE DISEASED POPULATION -- EPIDEMICS AND CONTROL." Volume III of the three-volume advanced treatise on Plant Pathology, Edited by J. G. Horsfall and A. E. Dimond. Published by Academic Press, New York-London. xiii + 675 pages. 1960. Price $22.

This final volume deals not with the intricacies of specific diseases and their effects on individual plants, but with the causes of the broad activities of plant disease agents, and with man's potential use of this knowledge in meeting his practical problems of plant-disease control.

In so doing, Volume 3 rounds out a learned but readable trilogy on plant pathology. It resulted from a task well done by editors Horsfall and Dimond, of the Connecticut Agricultural Experiment Station, who have brought together in these treatises the knowledge of many of the world's outstanding plant pathologists.

Volume 1 dealt with the diseased plant as an individual and Volume 2 with the methods of infection. Volume 3 is concerned with the populations of plants in relation to infection and control. It deals with the mass action of plant diseases and with the factors of inoculum, plant populations, and environment.

The concept of inoculum potential, characterized as energy potential, is carefully examined in early chapters by the editors, and by S. D. Garrett of the University of Cambridge. According to this concept, work is accomplished by movement of inoculum from source to successful infection of a new host, and frictional losses are due to factors tending to reduce the number of successful infections.

Inoculum dispersal, by insects and other animals, by air and water, and autonomously, is considered by various experts.

The application of knowledge of inoculum potential and dispersal to the practical considerations of plant-disease control takes up the last half of this 600-page book, with chapters on analyzing and forecasting disease epidemics, on useful quarantines, and on the classical cultural, chemical, and biological methods of disease control.

Contributors to this volume, in addition to the editors and Dr. Garrett, are: A. E. Muskett, of the Queen's University, Belfast; L. Broadbent, of the Rothamsted Experimental Station, England; C. T. Ingold, of the University of London; Harald Schrödter, of Germany's Agrarian Meteorological Research Station; J. E. van der Plank, of the Department of Agriculture, Union of South Africa; Paul E. Waggoner, of the Connecticut Agricultural Experiment Station; Ernst Gram, of the Statens Plantepatologische Forsog, Denmark; Russell B. Stevens, of George Washington University; W. A. Kreutzer, of the Shell Development Company, Modesto, Calif.; H. P. Burchfield, of the Boyce Thompson Institute for Plant Research, Yonkers; H. Darpoux, of the National Center of Agronomy Research, Versailles; and E. C. Stakman and J. J. Christensen, of the University of Minnesota. -- PAUL R. MILLER

"PLANT PATHOLOGY LABORATORY MANUAL," Second Edition, by J. P. Fulton, D. A. Slack, N. D. Fulton, J. L. Dale, M. J. Goode, and G. E. Templeton. Burgess Publishing Company, Minneapolis. iv + 95 pages. 1960. Price $3.

This new laboratory manual, designed to introduce the science of plant pathology to undergraduate college students, is commendable for both content and form.

Its subject matter includes discussions and examples of various types of bacterial and fungal diseases; of root knot and root lesion nematodes, and of the mosaic and yellows groups of viruses. The section, "Bacterial Diseases of Plants," for example, takes up soft rots, leaf spots, extensive blights, vascular wilts, and galls.

The manual also includes an introduction and a glossary; the former deals with such essentials as "Directions for Use of Microscope," "The Preparation of Temporary Mounts," and "Koch's Postulates."

The manual, of 8 1/2- by 11-inch pages size, is set up in a spiral binder so that the pages will lie flat without pressure. A readable type face was employed. The illustrations are large and distinct. -- PAUL R. MILLER